# ARCHIVES OF OPHTHALMOLOGY.

## THE CURATIVE VALUE OF ASPIRATION OF THE VITREOUS.

BY PROF. ZUR NEDDEN, DÜSSELDORF, GERMANY.

EXPERIMENTS in aspiration of the vitreous for removal of opacity as reported by Ford in 1890 and ten years later by Gradingo have remained unnoticed. This may be because the technic was not perfected, so that the dangers with which the method is frought seemed too great.

Also the experiments of Komoto in 1910 and of Elschnig in 1911 have, so far as I can discover in the literature of the subject, found no imitators. Both authors follow aspiration of the vitreous immediately with the injection of physiological saline solution, in the belief that thus the danger of retinal hemorrhage, which is great, might be averted. Elschnig later substituted air injection for the saline. In some cases his results were good, so far as bleeding and resultant opacity were concerned, but there was also serious inflammation.

In 1920 I introduced another method of aspiration of the vitreous which I had been studying for a long time. My experience has been published in numerous essays. Many authors have tested my experiments and have demonstrated the favorable result of aspiration in maladies of the eye. On the other hand, some failures have been reported. The unfortunate results I can only attribute to the technic of aspiration, and these mistakes will eventually be overcome.

During the past year I have had over 300 cases of vitreous aspiration, on which I am now reporting.

First I shall discuss the technic of aspiration.

My endeavor throughout will be to demonstrate that this interference is the simplest, gentlest possible, and yet the most effective.

Experiments on the eyes of dogs and clinical observation of the human eye have taught me that loss of the vitreous is speedily and spontaneously replaced—almost as quickly as the aqueous—so that substitution is immediately effected. And since the vitreous is usually liquified by the existence of the opacity, aspiration is accomplished without violent suction and without the need of dragging on the retina. The danger of detachment of the retina which might accompany suction seems to me to be small, especially when the amount of vitreous aspirated is relatively small.

Artificial substitution of saline solution for the vitreous is inexpedient, because the natural healing process is thus delayed; the physiological saline solution lacks all the healing power which is inherent in newly formed tissue, particularly the bactericidal and reabsorbent properties. And finally, saline injection in the vitreous is a dangerous interference because thereby may ensue irritation as well as infection. Interferences which avail nothing, but may do harm, are to be avoided.

In consideration of these reflections and facts I completely abandoned the artificial replacement of the opaque vitreous. And up to the present I have had no cause to regret my renunciation.

For the suction, I employ a record syringe[1] with specially prepared steel canulæ, sharpened like the lancet-shaped ones used by Elschnig. The tips must be accurately pointed and sharpened like lancet knives. I always test them on the test drum. On order to guard the point, the canulæ may be kept in a wooden case so that the sharpened tip touches nothing. There are twelve in the case, of varying thickness, with a lumen of from 0.3 to 0.8*mm*. They are sterilized by boiling, first having been cleansed with mercury-oxycyanide solution (1: 1500), since immersion in boiling water accomplishes only

[1] The entire instrumentarium for vitreous suction may be procured from F. L. Fischer in Freiburg, Windler in Berlin and other firms.

a superficial disinfection. The canulæ are put into little nickel containers for boiling, in which they are rigid and held firmly on all sides so that nothing can touch the point.

After disinfection, the lids and the conjunctival sac at the point of insertion are wiped with a tampon saturated with mercury oxycyanide. The puncture follows, either from below or from above outward 5 to 6*mm* from the limbus in a radiating direction, the globe being gently pressed against the inner orbital wall. Previously the bulbar conjunctiva has been pushed back by means of forceps toward the cornea. After removal of the needle it moves back to its original place and thus covers the scleral wound.

The canula is usually directed toward the center of the vitreous because there, according to our experience, the pathological change is most advanced. Too deep a penetration may be prevented by encircling the needle about 1½*cm* from the point. Suction should be carefully effected, without the exertion of force. Should any difficulty arise, it is best to stop and resume a few days later with a thicker canula. One must be careful to use the thinnest possible needle, because it penetrates more easily the great resistance of the sclera, and leaves a smaller wound, thus lessening the danger of amotio retinæ and infection. The largest needles I use are the intermediate ones of 0.5 to 0.6*mm* lumen. From 0.2 to 0.6*ccm* may be withdrawn according to the nature of the case. Later I shall discuss more minutely indications for aspiration of the vitreous.

It is possible that a laceration may occur in the scleral canal on the withdrawal of the needle from the incision—leading to unwelcome complications. To avoid this, I employ a split spatula and with it over the point of incision, press gently toward the eyeball which thus retains its shape. A bandage is worn for a few days after the operation.

Immediately after aspiration, the tension of the eyeball is greatly lessened. The bulbar conjunctiva becomes highly suffused and the anterior chamber seems a few minutes later to be slightly flattened, because traces of the liquid infiltrate back into the vitreous space. This disappears after a few hours. Hereupon a deepening of the anterior chamber usually manifests itself, which abates, after two or three days.

After aspiration, the eye is usually hyperopic, and lacks for every 0.1*ccm* of absorbed humor +1.6 diopter. On the following day, however, the original refraction returns, remaining stationary after the lapse of another few days.

In the case of an aphakic eye the needle may be inserted at the limbus and through the anterior chamber into the vitreous. To avoid an escape of the liquid the index finger should press the end of the syringe until the point is engaged in the vitreous. Should communication between the chamber and the vitreous follow the incision, paracentesis is almost synonymous with aspiration.

Elschnig and some other writers simplify the introduction of the canula by preceding it with a posterior sclerotomy, using the Graefe knife. The resultant wound is almost twice as large as with the lancet needle. But larger scleral wounds increase the danger of amotio retinæ which may occur weeks or months later and through shrinkage of the scleral wound a retinal laceration follows.

Bliedung claims that he can aspirate the vitreous by a posterior sclerotomy with a small knife, such as the Deutschmann employs for amotio retinæ. He allows the minutest possible quantity of the vitreous to trickle and refrains from doing an aspiration. There is no doubt that in this manner many vitreous opacities are cleared up. But the method would be of no value in cases of severe opacity.

The gentlest and at the same time the most effective method is and must remain incision with the lancet canula, because the resulting wound is very small and definite quantities of the vitreous can be drained. When the needles are carefully sharpened and prepared the puncture is made without difficulty so that we may avoid using a knife.

Aspiration is indicated for many internal eye conditions.

At first I shall concern myself with only ectogenic infections of the vitreous. Just one effect is to be striven for if the aspiration is to be done promptly and not too timidly. Best results are seen in cases of subacute inflammation of progressive infection. Twice daily I aspirate 0.5*ccm* thus assuring a speedy healing. In this way I have in many cases been able to save the eye and even have sometimes brought

about an appreciable improvement of vision. Blaisch and Basterra also report good results in many cases.

Also in ectogenic infections of the anterior eyeball, threatening to develop into panophthalmitis, aspiration of the vitreous may lead to cure as was shown by the case published by Erlanger.

The healing value of aspiration in intraocular infections depends on drawing from the eye a great number of germs and their poisons. Furthermore, with the replacement of the vitreous bactericidal substances of the blood will be transferred in the interior of the eye rendering the residual bacteria harmless. And very soon nutrition and resorption will be promoted.

Formerly there was an effort to treat intraocular infection by injection of iodoform into the vitreous. The curative effect cannot be the result of destroying pathogenic germs by disinfection, but only by the loss of vitreous caused by this procedure so that the results are similar to those following aspiration. The immediate germicidal effect of an antiseptic is of secondary importance in all suppurative processes. The organism heals of itself, through the agency of inherent healing power. The most practical treatment, therefore, is that by which the natural healing substances are attracted in increased quantity toward the focus of infection, as Bier has shown by his treatment with hyperemia. No treatment in intraocular infection is so effective as aspiration of the vitreous which may even aid when all other methods fail. The only other remedy which can be tried at the same time is the foreign protein treatment, which encourages the mobilizing and strengthening of the natural healing powers.

Noticeably better results are obtained by aspiration in cases of traumatic vitreous bleeding.

Obviously, however, one will not have recourse to aspiration at the moment of every vitreous hemorrhage resulting from an injury, since insignificant bleeding speedily absorbs spontaneously. In such cases it is best to wait for 8 or 10 days, as a conservative measure. If no noticeable clearing has taken place by that time there should be no delay in proceeding with the aspiration because there is danger of the organization of an effusion if the blood is allowed to remain

in the vitreous. Should connective tissue have formed, it is too late for aspiration. I have had frequent opportunities in cases sent me from other eye specialists for aspiration which were treated for months without benefit by conservative measures to observe this organization of the vitreous, and believe that a better result would have been secured by early aspiration. According to my experience, it is a great error of judgment to surrender such eyes to lasting blindness, through the omission of aspiration.

Many writers agree that the best results are obtained in aspiration in cases of traumatic vitreous hemorrhage. In such cases one can readily decide on aspiration since inflammation or similar complications are unlikely to occur. The first aspiration is usually 0.2*ccm*, for the second, after an interval of from two to three weeks, 0.5*ccm*. Occasionally a single aspiration suffices for the complete removal of the extravasation. In other cases the interference must be repeated several times. Even in the case of hemorrhagic opacity covering a period of years, not yet having formed a tissue, but of a mesh-like, powdery character, this method of treatment is permissible. I have found many such cases among my clinical material, and during many years of observation, I have seen no unfavorable results.

Greater care is demanded in cases of spontaneous than in those of traumatic effusion of the vitreous. Because there is danger here that following pressure exercised on the bulb at incision and after the great tension release connected with the aspiration there may be a recurrence of bleeding. But this danger must not be exaggerated. Experience shows that recurrent vitreous bleeding, which results from hemorrhage of the retinal vessels, infrequently follows immediately upon the aspiration, but usually a day or so later. This indicates that the venous blood of the retina is discharged with the release of pressure of the inner eye, while a swelling follows increased pressure. Aspiration in such cases does not encourage new hemorrhages. Conditions are exactly the contrary in cases of vitreous hemorrhage arising from arteriosclerosis. In such instances pressure release induces active hyperemia in the retinal vessels so that a new hemorrhage is more to be feared in connection with the aspiration. Never-

theless, even in such cases, the manifestation of hemorrhage after aspiration is no reason for avoiding the procedure since against such hemorrhage we are powerless. It is simply astonishing how a completely impregnated, perfectly opaque vitreous is cleared as early as the day following aspiration.

It seems to me quite remarkable in recurrent vitreous hemorrhage, in case no spontaneous resorption follows, to find in spite of all relapses, indications for vitreous aspiration. I have a notion that it is helpful in cases of tuberculous processes, where there has been retinal hemorrhage. And I believe that the hyperemia following aspiration, which is, according to Bier, one of the most effective means of fighting local tuberculous processes, also favorably affects the tuberculous retinal focus.

Naturally all vitreous hemorrhages are not to be cured in this way, since there are cases in which all therapeutic measures are contra-indicated. But I have never had a case in which a careful procedure has given rise to any mishap. One must be warned, however, to handle the slender canula properly, and never to absorb more than 0.2*ccm.*

Not only in hemorrhagic opacity of the vitreous but also in its resultant iridochoroiditis, aspiration may prove valuable.

In this group the selection of cases should be exercised with even greater care than in those of spontaneous hemorrhage. A few cases are indicated in literature in which aspiration resulted harmfully. This can only be because the technic was faulty or that aspiration was not indicated in the particular instance.

The most important presupposition is that the eye must be absolutely free from irritation. Inflammation must therefore have greatly lessened. Also I select only cases in which the vision is much impaired in consequence of the vitreous opacity.

Complete fluidity of the vitreous sometimes noted in this condition demands, as a rule, the thinnest needle of 0.3*mm* lumen. This has the advantage that incision with the lancet needle is easily performed so that heavy pressure, which such eyes resent, may be avoided. The resultant scleral wound is very minute. Particular precautions against inflammation are demanded by both types of case. If one

is contented with the release of a small quantity, 0.2*ccm* at the most, no complications are to be feared. But repetition must not be undertaken too soon—usually not until after three weeks or more.

Success depends, as in the case of vitreous hemorrhage, partly on the mechanical removal of the opacity, partly on the progress of resorption following aspiration. Improvement is not immediately noticeable after the interference, but is progressive as time goes on.

In spite of all precautionary measures, occasional irritative conditions may arise, since it is impossible to judge with certainty in the case of a chronic iridocyclitis whether it is suited for aspiration. In such cases a second aspiration is not resorted to. But harm cannot result, if precautionary measures have been taken. Some eyes submit so easily to aspiration that there is not the slightest danger of bad results. In these cases the procedure influences the fundamental trouble which caused the opaque vitreous by improving nutrition.

So too vitreous aspiration is indicated and sometimes succeeds in cases of diffuse choroiditis. As there is no opacity in such cases, one hesitates to aspirate. But should vision be much impaired, indicative of continuous deterioration, I consider this method permissible provided all other procedures have been without result. The procedure is accompanied by no technical difficulties because the vitreous is fluid and dangerous inflammatory symptoms are lacking.

In recent years vitreous aspiration has been practiced in other eye diseases. Rosenstein has availed herself of it in occasional cases of neuritic atrophy of the optic nerve with good result. Here, too, better nourishment and increased resorption explain the improvement.

We may even consider vitreous aspiration in embolism of the central artery of the retina, so often treated by repeated paracenteses. The recovery in these cases depends upon the dilation of the retinal arteries which follows this procedure and the resulting reduced ocular pressure. This effect is greater after aspiration than after paracentesis.

In many cases of traumatic glaucoma vitreous aspiration is of great value. I am thinking of cases of pressure-increase

following a contusion of the eyeball and usually connected with subluxation of the lens.

Many writers take the viewpoint that tension increase in these cases is due to pressure on the lens, by which pressure on the ciliary body is effected. I am not satisfied with this suggestion. Before I practiced vitreous aspiration, I once made a microscopic examination of an eyeball in a case of this kind immediately after enucleation and discovered that the displaced lens was not in contact with the ciliary body. On the other hand I found in spite of the youth of the patient that the central part of the vitreous was fluid and that the peripheric parts were inspissated and adhered to the retina on all sides as a viscous, gelatinous mass. This last is, in my opinion, the real cause for the elevation of tension, which in turn impedes resorption.

Experience has shown me that injury brings about a serious change in the vitreous, resulting in the rise of tension. In cases of traumatic glaucoma in which I have performed vitreous aspiration the peripheral portion of the vitreous could not be aspirated on account of its viscosity, while the center was watery like the aqueous.

I have had permanent cures in four cases of this kind, from aspiration, in two of which a single operation was sufficient.

Also in a case of spontaneous subluxation of the lens complicated by glaucoma, aspiration effected a cure, requiring four repetitions while the iridectomy which I had done before was without result. After six years' observation, pressure has remained normal although the subluxation of the lens remained unchanged, so that the upper part of the pupil is aphakic.

Therefore, lens displacement cannot be the reason for pressure-increase, but rather, the glaucoma is the result of tremendous alteration of the vitreous which, as I can demonstrate by aspiration, is thickened at the periphery and liquified at the center.

In all these cases improvement following aspiration is undoubted, since the malnutrition, inevitable from the serious alteration of the vitreous, and especially from the thickened periphery, is overcome by the aspiration which restores resorption.

Also in a case of hemorrhagic glaucoma which I have described elsewhere I was able to verify favorable results after aspiration. But I am far from declaring this method to be best in all cases of hemorrhagic glaucoma, because with traumatic glaucoma as well as with spontaneous, lens displacement is not accompanied with conditions to make it effective. Further clinical experiment will perhaps direct us to the correct method.

There is another form of glaucoma in which I do not advise aspiration, because it is to be borne in mind that with the renewal of the vitreous after aspiration the albumen content as well as the liquid is increased. This might easily, in the case of an eye suffering from glaucoma or inclined to it, cause injury. But Bufili and some other writers saw in cases of glaucoma where the anterior chamber was quite flat good results from aspiration. A final decision may only be reached after further research.

In cases of high myopia, aspiration is to be condemned, from danger of amotio retinæ which is greater in extreme nearsightedness.

But for the rest, by my method amotio retinæ is not to be feared. In more than 300 cases of aspiration which I have performed as well as among the numerous cases of Rosenstein there were none in which this complication resulted from the operation. More likely is the detachment of the choroid as is observed after cataract operations. This is not serious as spontaneous recovery takes place.

So we see that the diseases in which vitreous aspiration is indicated are very various. Yet the cure in all cases is the same, namely, a promotion of resorption induced by the renewal of the vitreous. The mechanical removal of the vitreous opacities adds another healing factor just as in infectious processes a great number of germs and their poisons are eliminated by removal and bactericidal substances of the blood are made available.

By a careful selection of cases and with the observation of all technical rules, vitreous aspiration may be of service in numerous serious eye diseases where other treatment has failed.

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# DETACHMENT OF THE ZONULA LAMELLA.

By Dr. S. J. MEYER, Chicago, Ill.

Honorary Assistant, The German University Eye Clinic, Prague, Director, Prof. Dr. A. ELSCHNIG.

(*With two illustrations on Text-Plate* V.)

SLIT lamp examination of the anterior part of the eyeball has resulted in a newly revived interest in and study of the zonula lamella.

The zonula lamella at first was believed to be present only in the equatorial part of the lens, and had been observed clinically only in pathological cases such as subluxation of the lens or following trauma.

However, Elschnig in 1922 was the first to publish findings in three eyes from two glass blowers in which he believed he saw a detachment of the zonula lamella throughout the entire anterior capsule. The findings in his first case were as follows: "On the anterior lens surface there lies a polygonal, thin, transparent membrane, with gold glistening margins, which vibrated upon any eye movement. The margins were rolled lightly anteriorly, especially above, so that the entire formation appears to be attached to the lens only in its middle part and extends concavely towards the anterior chamber." The second case revealed in the right eye a transparent membrane about 3*mm* wide lying on the anterior lens surface, which fuses above into the anterior lens surface and is rolled up towards the anterior chamber below near the pupillary margin, and floats slightly upon any movement of the eyeball. The left eye in this case revealed a glass-like membrane covered with single pigment granules on the anterior lens capsule, the lateral and

inferior margins slightly folded and curled posteriorly, and appearing to be attached to the lens capsule only in the upper part.

Elschnig believes that the membrane seen was the zonula lamella separated from the entire anterior lens capsule, and concludes that the zonula lamella protects and overlies the entire anterior lens capsule, while former investigators believed that the zonula lamella was present only in the equatorial part of the lens capsule.

Kubik in 1922 examined 63 glass blowers ranging in age from 18 to 61 years, all of whom had been in contact with intense furnace heat since they were 14 years old, and in five cases observed a rent in the most anterior layer of the lens capsule which we conceive as the zonula lamella.

Elschnig in 1926 also reported the case of a blacksmith who had a detachment of the zonula lamella, which was the first case ever reported in an individual whose occupation was other than that of a glass blower. The slit lamp examination in this case revealed in the right eye an extremely delicate membrane, folded in part, covered with fine pigment, lying medially in the anterior chamber and extending to the posterior corneal surface, which appears to have been torn off from above and then rolled downwards. Temporally the membrane fuses directly into the lens surface and one can only follow single transparent strands up to the lens margin. The left eye revealed a very similar picture. He stated that this case appeared to confirm his view regarding the etiology, that it is not the light rays, but the activity of the heat rays which lead to a wrinkling and detachment of the zonula lamella. The article is summarized with the statement "that in reality a rupture and detachment of the zonula lamella in glass blowers is produced by the radiation of the heat in such a way that at first without any change of integrity of the lens, a change of elasticity, then rupture, and finally detachment of the zonula lamella takes place, and that the lens opacity is entirely independent of it."

Weill and Yole Levy in 1926 also reported a case of bilateral detachment of the zonula lamella which occurred in a blacksmith who had worked near a very hot fire for many years. The slip lamp examination revealed a hexagonal figure situated

ILLUSTRATING DR. S. J. MEYER'S ARTICLE, "DETACHMENT OF THE ZONULA LAMELLA."

Temporal Nasal

FIG. 1.—Right Eye.

Nasal Temporal

FIG. 2.—Left Eye.

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in the center of the pupil with its margins extending into the anterior chamber. The margins are sharp, glistening and silvery, somewhat curled and float easily upon any eye movement. The membrane is structureless, transparent and gray. Centrally it lies upon the anterior lens capsule into which it fuses. The picture made the impression that this membrane had become detached from the lens capsule. It seems apparent that the authors at that time were not aware of Elschnig's previous findings or his theory regarding the etiology.

Stein in 1926 recorded a "Detachment of the Zonula Lamella in a Case of Bilateral Spontaneous Lens Subluxation," in which the slit lamp examination revealed the detached zonula lamella midway between the upper pupillary border and the superior margin of the subluxated lens.

Jess in 1926 reported a case of "Detachment of the Zonula Lamella" which was observed histologically in an eye enucleated in a man 65 years old on account of a large corneal staphyloma following an ulcus serpens. The lens was luxated anteriorly, and there was a delicate hollow lamella detached from the ciliary body. Microscopic sections showed that this consisted of a thin hyaloid membrane which stained the same as the lens capsule. Jess believes that the zonula lamella is only on the equatorial part of the lens in spite of the fact that Elschnig, Schnyder, etc., believe it to extend over the entire lens capsule.

Rotter observed in 1925, following the examination of 133 glass blowers, a case showing a typical detachment of the zonula as noted by Elschnig in 1922. In 1926 he reported a case which revealed a particular form of zonula lamella detachment. This appeared in the anterior chamber as a transversely oval, plicated band, lying upon the anterior lens surface nasally below and temporally above, which formed a free arc between these points. This observer agrees with Elschnig that the condition is due to heat radiation. Compared with the first case of my publication it seems that Rotter mistook the picture seen in his patient, because he believes that the zonula lamella in the marginal part is still attached, and only detached in the middle. According to our findings it seems more probable that the

detachment of the zonula lamella has its origin from the lens margin, and that the anterior pole of the lens is still covered with the lens capsule.

Elschnig believes it possible that the building of folds in the anterior shagreen substrate as observed by Vogt (P. 160) is identical with the zonula lamella, and that in another case recorded (P. 204), the described network of threads has the same origin. Vogt is also of the opinion that there is some similarity or relation between his findings and that of Elschnig. The depicted separation of the zonula lamella may also have some significance regarding the criticism of the pathogenesis of glass blowers' cataract, which was developed by Wicke. It is not good reasoning that the detachment of the zonula lamella is due to the action of light, although it might easily be accounted for by the action of heat radiation. This is in conformity with Vogt's theory regarding the pathogenesis of cataract in glass blowers.

Wollenberg in 1926 reported the observation of an annular membrane formation with curled edges on the anterior lens capsule of a woman with a myopia of 11 D., which he believed was attributable to the rubbing of the pupil on the anterior lens capsule during pupillary movements. This may have been a zonula lamella very similar to the findings noted by Vogt in his cases.

Cords in 1926 examined the eyes of a large number of men employed in occupations where their eyes and faces had been subjected to intense heat and light rays over a long number of years, in order to ascertain the percentage of cataract formation prevalent. Amongst these cases he found detachments of the zonula lamella in two cases. The findings in the first case were described as follows: left eye, a central corneal opacity accompanied by a detachment of the zonula lamella. In front of the center of the lens there floats freely a fine transparent membrane connected with the anterior lens surface. The membrane is about *2mm* wide and the lens shagreen is somewhat spotted. The second case revealed a detachment of the zonula lamella in both eyes. In the right eye one could see a delicate membrane floating, and hanging down over the entire lens from its attachment on the temporal side. It was about *5mm* wide. The left eye revealed a similar

membrane, somewhat more delicate and smaller. The remaining parts of both lenses were entirely normal.

Schnyder in 1926 reported the results of his examinations regarding the professional cataract as noted in iron workers and glass blowers who are subjected to the action of intense heat and light rays for long periods of time. In all, he examined 32 iron workers who were continually subjected to a temperature of 1300–1500° C. Amongst the cases numbered 24–32 he found that nine of the eighteen eyes revealed changes in the capsule lamella similar to the changes noted in our cases. The nine eyes revealed a lamellar separation of the anterior lens capsule, and the findings were identical with the changes first observed by Elschnig, and described by him as a "Detachment of the Zonula Lamella."

Alling in 1927 described seven cases of "Exfoliation of the Lens Capsule." In attempting to account for the etiology he states that "The obvious explanation of these cases is that a superficial lamella of the anterior capsule has degenerated and become friable so that the normal movements of the iris in the contraction and dilatation of the pupil have denuded the rest of the capsule which remains clear, leaving the slightly opaque disc in the very center." This explanation is essentially similar to that given by Wollenberg in 1926.

The majority of the cases recorded in the recent literature have occurred in glass blowers, whose eyes and faces are continuously subjected to intense heat over long periods of time.

The first case I have to report occurred in a blacksmith, the second in a woman following an intracapsular cataract extraction.

Franz H., age 48, blacksmith by occupation, was admitted to the clinic January 1, 1927. He stated that for the past two years the vision in his left eye was foggy and had been rapidly failing. For the last two months he had no vision in the left eye. He had also noticed a reduction in vision in the right eye during the previous two years. Formerly had good vision, but had worn glasses during the past three years for reading. The family history was negative.

Vision R. E. = $\frac{6}{10}$?, not improved with glasses. Vision L. E. = candle at 6 meters. Projection correct.

Both lacrimal sacs patent. Lids, eye position and movements normal. Tension with Schiötz tonometer R = ½, L. = ½. The right pupil was 4*mm* in diameter, the left 3*mm*. Good reaction to light. The anterior chambers are somewhat shallow, the left more than the right.

Examination of right anterior lens capsule with the loupe revealed that the central part is about one-fifth the size of the lens diameter, irregularly shagreened, while towards the periphery there is a gradually diminishing opacity which appears as two small winding membranes in the anterior chamber.

Slit-lamp examination of the right eye revealed the fact that the zonula lamella appears to be detached from the lens capsule on all sides except the temporal part. The upper half appears to be turned forward and hangs like an ampulla or bubble which extends into the anterior third of the anterior chamber. The inner part is curled together above, and projects medially to the part from the upper half, which is twisted under and covered, also protruding far into the anterior chamber. The structure is perfectly homogeneous, except that it appears to be covered with fine pigment. There is no membrane to differentiate on the lens capsule at the center of the anterior lens cortex except a moderately thickened opacity of the lens present there. This area amounts to about 1*mm*, and about this region the zonula lamella appears to be loosened. Only towards the temporal side is the zonula lamella attached to the lens capsule. At 9 o'clock there are three radiating bands which may conform to a plication or twisting of the zonula lamella and disappear under the iris.

The posterior corticalis is equally dense all over, irregularly shagreened and contains fine opacities.

The slit-lamp picture is clearly indicated in Fig. 1.

The left anterior lens corticalis reveals a peculiar sector formation, and is irregularly clouded, with two definite circular opacities through its entire thickness. There is no essential change in the picture towards the nucleus. The posterior capsule cannot be seen.

On January 12, 1927, after venepuncture of 150*cc* of blood, Prof. Elschnig performed a simple Stanculeanu-Török intracapsular extraction of the left lens, which was followed by a normal convalescence and a vision of ⅔ with the proper lens correction.

There can be no doubt that the transparent membrane situated in the pupillary area and extending sideways into the outer layer of the lens capsule, is itself a part of the lens

capsule. As the lens capsule itself can anatomically be separated into layers chemically or by maceration, then the separated membrane probably cannot be other than the zonula lamella of the lens capsule. So far as one can determine conclusions regarding the normal anatomy from pathological changes, it can also lead to the conclusion that the zonula lamella surrounds the entire lens capsule, or at least the entire anterior part, a theory in vogue at our clinic. Clinically, however, up to the time of Elschnig's first publication, the zonula lamella had only been seen isolated in the equatorial region of the lens in eyes examined pathologically on account of severe irido-cyclitis.

The second case is that of a woman, J. C., age 55, white, who first came to our clinic on March 31, 1926. At this time she gave a history of having had a very sore, red right eye for the previous six weeks, which was treated by her physician as an iritis by means of atropine instillations and potassium iodide internally. The condition had slowly, but gradually improved, and she then complained of gradually failing vision in the left eye. No previous inflammation of the left eye had occurred at any time.

The past history revealed that the patient had had nine pregnancies and no abortions, but only three children were living, most of them having died in very early infancy. There had been no rheumatism or previous attacks of tonsillitis.

The urine was negative for albumin, sugar and indican. The blood pressure was $\frac{1}{1}\frac{8}{0}\frac{8}{0}$.

Vision of R. E. = $\frac{6}{24}$, without improvement by glasses. Vision of L. E. = fingers at one meter with correct projection.

Examination of right eye revealed evidences of a subsiding iridocyclitis such as a cloudy corneal epithelium, a slight tongue shaped corneal opacity between 2—3 o'clock with superficial blood vessels entering from above, and precipitates on the posterior corneal surface. The iris markings were somewhat obscured. The pupil was irregular with a posterior synechia at 7 o'clock. There were pigment particles present on the anterior lens surface. The lens revealed irregular minute opacities both in the anterior and posterior corticalis. The fundus was negative.

Examination of the left eye revealed an absence of injection. The cornea contained slightly pigmented precipitates on the posterior surface. The pupil was bean

shaped with a posterior synechia at 6 o'clock. Pigment was present on the anterior lens capsule near the pupillary border. The lens revealed punctiform opacities in the anterior cortex with a large leaf shaped opacity.

Tension with Schiötz tonometer R. E. = $\frac{1}{3}$, L. E. = $\frac{1}{3}$.

Dental examination revealed three carious teeth which were immediately extracted.

On April 8, 1926, the blood Wassermann was found to be positive.

The patient received local eye treatment to the right eye consisting of heat, scopolamine and dionin. This was accompanied by six intravenous salvarsan injections and two series of mercury rubs. Under this treatment the right eye cleared up fairly well, and patient was discharged April 21, 1926, and told to return later for cataract extraction of the left lens, after she had received more anti-luetic treatment. Patient reported at irregular intervals and was again admitted to the clinic on January 3, 1927, at which time vision of the left eye was fingers at 20*cm* only on temporal side, with a candle still visible at 6 meters, but a bad sense of projection on the nasal side. The blood Wassermann was again found to be positive and she was immediately put on anti-luetic treatment.

On January 11, 1927, the left lens was extracted by Assistant Dr. Hermann Elschnig by means of the Stanculeanu-Török intracapsular method with an iridectomy. The capsule was intact and recovery was uneventful.

January 25, 1927, fourteen days after extraction, vision of left eye was $\frac{3}{6}$ with +10.0S. ⌒ +5.0 cyl. × 150°. Javal = 150° 6.0 D.

Examination of the operated eye at this time revealed practically no injection. The upper part of the cornea was still somewhat cloudy. The anterior chamber was deep. The aqueous contained some pigment. The iris revealed a poor stroma, and exudate on the pupillary margin with a broad parallel coloboma extending upward. The pupil was central, 6*mm* in dia., and did not react to light. In the pupillary region temporally and inferiorly, there could be seen a thin brown membrane protruding into the anterior chamber, which appeared as a zonula lamella. The membrane appears to lie upon the vitreous with which it moves. The anterior margin of the lamella is free. The anterior vitreous body membrane is intact. The vitreous extends far into the anterior chamber, and its margins are richly pigmented with brown pigment. There are also coarse, floating vitreous opacities.

Ophthalmoscopic examination revealed the left papilla to be round, somewhat unsharply defined, slightly pale and

its arteries thin. Temporally from the papilla in the macular region and laterally but below the upper temporal artery, there is a small group of sharply defined white flecks. Peripherally and superiorly, visible only through the coloboma, the retina is gray, atrophic, the arteries are sclerotic and pigmentation is present. Below the papilla there are also chorioiditic foci.

Slit-lamp examination of the left eye revealed a glistening membrane which is moderately thick and contains pigment, extending forward from under the lateral pupillary margin into the anterior chamber. The anterior margin of the membrane runs in an apparent arc, which ends outwardly as a sharp point, and similarly below. Superiorly the membrane lies on the flat part of the vitreous hernia. Anterior to the concave surface of the film there is no vitreous. The lower pointed end floats freely in the aqueous. The anterior margin is doubly contoured and glistening. This thin, transparent, glistening membrane can be nothing other than the zonula lamella which is still attached to the zonular fibers at the temporal margin.

Fig. 2 gives a clear picture of the transparent, glistening membrane with its origin under the temporal and lower border of the pupillary margin and its medial end extending anteriorly into the anterior chamber.

The older anatomists Babuchin, Arnold (cited by Becker, *v. Gr. S. Hand.*, 1 Aufl.), had already recognized that the lens capsule was not a homogeneous membrane, but possessed a stratification. Arnold believed that the anterior capsule was composed of two layers (or even three), the outer belonging to the Zonula of Zinn, the inner to the lens capsule.

Berger by means of his maceration experiments on the lens capsule was first to recognize the outer lamella or layer, which he designated the zonula lamella of the lens capsule. Berger found that by unraveling the macerated lens capsule with potash of permanganate, the outer layer revealed a different physical property than the deeper or inner layer, in that in contrast to the more irregular zigzag marginal rents, it possessed straight torn margins. He thought this property was due to a greater elasticity of the zonula lamella. He also observed a spontaneous partial detachment in an unmacerated capsule. His theory was that the zonula lamella, like the lens capsule, was of mesodermal origin; a theory

which was later disproved by numerous more exact observations, which showed that the lens capsule was a separation product of the lens epithelium, and is, therefore, a similar ectodermal tissue (Rabl, Ritter, Schwalbe).

The later, apparently numerous anatomical and pathological investigations regarding the suspension apparatus of the lens resulted in practically nothing new regarding the true zonula lamella or the finer structure of the zonular fibers on the capsule. Contrary to Berger's findings, the later investigators were unable to confirm the presence of a zonula lamella.

Salzmann reports in his *Anatom. Studie über die Zonula ciliaris usw.*, that the zonular fibers fuse into the capsule and are limited to the equatorial region only, without a lamellar continuation. Hess (*Gr. S. Hand.*, 2 Aufl.) and many others who have worked on this subject, stress most of their investigations upon the route and origin of the fibers, and upon the distribution of the single fiber bundles on the anterior, middle and posterior insertion lines on the lens capsule, without considering the possibility that the further insertion of the radiating bands could be a zonula lamella continuing over the entire anterior capsule.

Meesmann, who further undertook the investigation with the aid of the slit lamp on fresh equatorially sectioned human and animal eyes was unable to make any further accurate determinations with relation to the zonula lamella. He plainly observed as an evidence of beginning post-mortem change, an equatorial lifting up of the zonula lamella, and further, that often after injections with Zenker's solution, a small zone concentric to the lens margin first became opaque, which after some length of time was lifted up from the surrounding part.

Busacca in 1927 by means of the silver impregnation method of Bielschowsky was able to establish the existence of the zonular fiber bundles, which contrary to the general assumption penetrate into the lens capsule, where they can unquestionably be followed inside the outer two-thirds of the capsule, besides the bundles of fibers already written about by the older authors Berger and Retzius, and which spread out on the outer surface of the capsule. Other bundle

fibers also penetrate inside the capsule, but remain in the outermost layers. He also found that this impregnation method revealed the existence of a thin layer or lamella extending around the entire lens capsule, which stains black in contrast to the capsule and the zonular fibers, which stain red-violet when gold chloride solution is used. In the equatorial and peri-equatorial zone the structure of this layer is most prominent, and distinctly appears here to be made up of bundles of wave-like fibers. Zonular fibers lie on it, while the large zonula fiber bundles which were seen penetrating into the capsule, extend through this layer, and only a few fibers, from the outer fibers of the bundle, spread out upon it. This layer which has a definite development and on which the zonular fibers lie, becomes thinner the further we go from the peri-equatorial zone; the wave-like winding of the fibers becomes less and more unclear, until the layer takes on the appearance of a thin cuticular structure, and is contrasted by its black stain. This picture is also present in the zone of the lens capsule which corresponds to the pupillary field. We believe this layer to be the zonula lamella, although Busacca takes Schnyder's viewpoint which denies that the zonula lamella extends over the entire lens capsule, and states that the clinical findings are not a detachment of the zonula lamella but a lamellar separation of the superficial layers of the lens capsule.

The question regarding the development must for the present remain undetermined. One can only see the zonula lamella in the uppermost layer of the lens capsule, which in consequence of the exclusive resulting radiation of the zonular fibers can become detached in its equatorial part under various circumstances. On the other hand, however, there is Meesmann's assumption that the zonula lamella is a part of the zonular fibers, which must be given careful consideration.

Only very few of the newer investigators still adhere to a mesodermal origin for the zonula. Most of the investigators maintain it to be an ectodermal structure, and in reality after Carlini, Mawas and Magitot, a differentiation product of the anterior capsule of the membrana vasculosa, where the zonular fibers insert next to the capillaries (Carlini); not only single fibers, but a lamella can be differentiated, which is

later left attached to the lens capsule after the membrana vasculosa disappears. After Beauvieux the lens consists of (1) an inner part, which can be seen as a production of the epithelial cells of the lens, on which lie the rests of the embryonal capsula vasculosa lentis; (2) as an epithelium, externally from this, which is only met with in the sphere of the equatorial zonular fibers, derived from these fibers and also of ectodermal nature.

The cases reported and the ensuing discussion are given with the hope that interest and study of the obscure zonula lamella may be further stimulated; also that routine post-operative slit lamp examination following intracapsular cataract extraction may lead to a more frequent observance of the condition presented, especially in eyes where the extraction has been combined with an iridectomy, and, therefore, the remaining zonular fibers could lie visible in the ciliary region of the iris coloboma.

I also wish to take this opportunity to thank Prof. Elschnig for his instigation, corroboration and permission to publish the cases.

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# THE SIGNIFICANCE OF THE CILIARY EPITHELIUM IN THE ABSORPTION OF VITREOUS HEMORRHAGES.

By Prof. J. MELLER, Vienna.

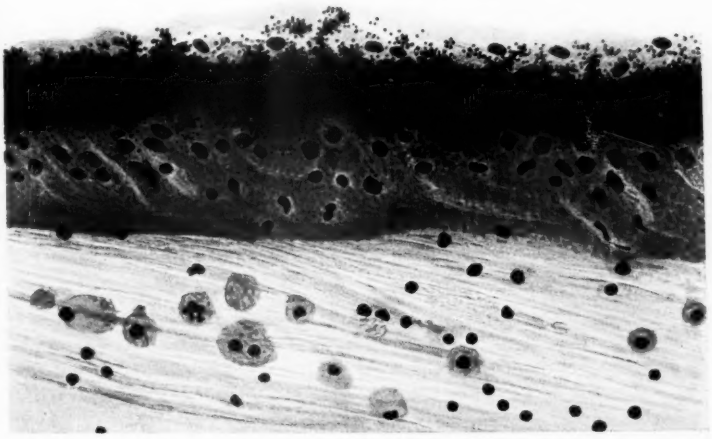
*(With one illustration in colors on Text-Plate VI.)*

OUR knowledge regarding hemorrhages in the vitreous is still fragmentary. Clinical experience teaches that, as a rule, hemorrhages in the vitreous are absorbed slowly and that even copious bleedings may disappear completely in the course of time. The most favorable conditions obtain in otherwise healthy eyes, in which the hemorrhages are due to a trauma which affects only a circumscribed spot, without involving the other tissues of the eye, or, where a spontaneous bleeding into the vitreous occurred in circumscribed phlebitis.

On the other hand, if the trauma caused grave injuries to the tissues and necroses in the internal ocular layers, as may happen after severe contusions, or where the internal layers are impaired from chronic inflammation, the absorption is hampered, remains inadequate, and in its place we have organization of the effusion and subsequent induration. Amongst injuries which decrease and finally obliterate the absortive power, we must give especial attention to the vitreous hemorrhages themselves, because through the repeated occurrence of these hemorrhages the absorption becomes gradually slower, and finally ceases entirely.

The interval between the onset of the hemorrhage and the first manifestations of the absorption varies. In one case the changes may be noted after a few days, while in other cases the condition remains unchanged for weeks, so that one might give up all hope that the vitreous would ever become clear again.

ILLUSTRATING PROF. MELLER'S ARTICLE ON "THE SIGNIFICANCE OF THE CILIARY EPITHELIUM IN THE ABSORPTION OF VITREOUS HEMORRHAGES."



Showing changes in the protoplasm of the epithelial cells in the inner unpigmented layer of the pars plana of the ciliary body.

There is also a difference in the progress of absorption. In most instances the opacity of the vitreous disappears gradually, while in others no improvement is noticeable for weeks, or months, and then, suddenly, the results of copious hemorrhages will disappear in a surprisingly brief time.

I want to describe one case of vitreous hemorrhage, which is instructive for several reasons:—

A. S., male, aged 44 years; during the past six years gradual decrease of vision in the left eye, due to a gray cataract, the cause of which could not be discovered. At the iridectomy, done for the extraction of the cataract, a completely liquified fluid vitreous trickled out of the wound. During the removal of the lens with a loop more of the vitreous was lost. The day following operation the chamber was deep, the aqueous humor clear. On the fourth day after operation, the conjunctival flap was suffused, and the anterior chamber was filled with blood. During the following days the blood in the anterior chamber was absorbed, but five days later the chamber was again filled with fresh blood.

When, a fortnight after operation, the patient left the hospital, the aqueous humor was still tinged with blood and only scant red reflex emanated from the fundus, undoubtedly due to hemorrhage into the vitreous. Vision was reduced to hand movements. In the following weeks, while the hemorrhages into the vitreous body continued, vision dropped to the perception of light in 3*m;* with defective projection nasally. As, even in the sixth week after operation, vision had not improved, the vitreous hemorrhage showed not the slightest indication of absorption, and the eye remained soft and injected. Enucleation of the eye was recommended, which the patient refused. Five months later we were notified by a colleague that the eye was pale with vestiges of blood in the anterior chamber, the vitreous body was so opaque that no details could be distinguished, but vision had improved to $\frac{1}{10}$. The patient appeared in our clinic 16½ months after removal of the cataract. The central parts of the vitreous had cleared up completely, so that he could read with cataract glasses $\frac{1}{12}$ and Jaeger 1. But in the periphery, nasally, temporally and inferiorly the red light was still dull.

This case shows that (1) the vitreous, completely filled with blood, had cleared up surprisingly well after a consider-

able amount of vitreous humor had been lost during the operation; (2) In the sixth week after the onset of the hemorrhages no absorption had taken place; (3) Probably only after several months had absorption progressed quite satisfactorily.

It is well known that the blood extravasated into the vitreous, owing to influences with which we are not yet acquainted, varies in its behavior: in some eyes it does not change its morphological character for months, and the red blood corpuscles are not distinguishable from those of fresh hemorrhage, while in others a rapid decomposition of the elements is noticeable, the blood-pigment is extracted, etc.

The anatomical processes accompanying hemorrhage have been described by E. Fuchs (*Graefe's Archiv*, Vol. 99, 1919). The result of his investigations regarding the question "How is the blood carried off?" is quite meagre. For the cells studied by Fuchs in eyes with hemorrhages in the vitreous consist of derivatives of the pigment epithelium, mobilized and migrated into the vitreous, and phagocytes which are too small in numbers to play any important part. The phagocytosis occurring in vitreous hemorrhages is small in comparison with that occurring in hemorrhages in other tissues. This fact is striking for, during the examination of eyes where a hemorrhage has occurred into the anterior chamber, the surface of the iris was found covered with a whole layer of phagocytes, while there were only a very few in the vitreous body. On this basis, Fuchs tries to explain that pigmentation is frequently absent after hemorrhages. Therefore, in his opinion, owing to the insignificant rôle played by the phagocytes, complete absorption can result only when hemorrhages are slight, for phagocytosis is not able to carry off larger amounts of blood. This leads to the following question: "How then does the absorption of the blood come about? We may suggest three different processes, which partly take place simultaneously. (1) By the result of hemolysis, the contents of the erythrocytes extravasate and, in a dissolved form, pass through the vitreous to the excretory ducts, through which they flow out with the lymph; the extracted stromata of the erythrocytes disappear by solution. (2) After their extravasation, the contents of the erythrocytes are precipitated as granules, which afterwards

become again dissolved and are carried off with the ocular fluids via the normal ducts.

"The late solution of the granules is proved by the fact that in cases of more than five weeks' standing they are no longer discernible, though in many of such cases they were certainly present before."

The third point mentioned by Fuchs, namely, the growth of connective tissue, lies outside the scope of our investigation.

In brief, Fuchs believes that in the process of absorption of vitreous hemorrhages a solution of the extravasated blood takes place which then leaves the eye by the usual excretory channels. If this were really the case, one might assume that, owing to the important rôle played by the anterior chamber (iris, Schlemm's canal), in the removal of the intraocular fluid, one would notice more of it in this space. Fuchs has also been struck by the fact that, in spite of the frequent occurrence of the hemolysis in the vitreous, this is rarely observed in the anterior chamber. He advances the following explanation: hemolysis takes place only gradually, the released hemoglobin is precipitated in the form of granules, so that the amount of dissolved hemoglobin is too small to tinge the aqueous red on passage into the anterior chamber. Besides, the anterior limiting layer of the vitreous possibly constitutes an impediment to the diffusion of the hemoglobin into the aqueous humor, which would be quite understandable if we take into consideration the size of the hemoglobin molecule. Also the discoloration of the iris into a brownish-green is observable only occasionally in vitreous hemorrhages. The erythrocytes may even disappear without the occurrence of a hemolysis.

In Lauber's article (*Graefe-Saemische Handbuch*, 1921) on the vitreous body, published after the appearance of Fuchs' paper, we likewise find no explanation for the process of absorption. Except the remark that occasionally dissolved hemoglobin is found diffused in the vitreous and is then removed from the eye through the current of the intraocular fluids, Lauber has nothing to say regarding the processes which lead to the disappearance of hemorrhages in the vitreous.

He notes that, besides the hemolysis, the blood corpuscles undergo other changes and that swelling and disintegration

have been observed; and adds that, in the absorption of blood, these processes play only a minor rôle; but he fails to explain in what manner this débris is removed. Of fibrin he says that, in case it is not absorbed early, it is transformed into an amorphous, structureless mass, the absorption of which would require considerable time. But Lauber does not tell us anything about the character of early absorption, nor how the absorption of this amorphous mass would come to pass after a considerable time.

After all, the healthy eye seems able to digest blood without being dependent upon its removal after solution by way of the intraocular circulation.

Lauber himself mentions a case of Collins, in which a total vitreous hemorrhage, following an injury, disappeared completely after six days, and in one of his own cases, a boy, 11 years of age, the vitreous of the left eye, after an injury, was so congested with blood that perception of light was reduced to a minimum; but after a fortnight the vitreous was again so transparent that vision was 1, and after another two weeks no vestige of the hemorrhage was discernible. Lauber reiterates that only an *otherwise normal eye* is able to absorb a hemorrhage so quickly and thoroughly. Statements like "if affections of the retinal vessels or inflammations of the inner tunics of the eyeball are present, which retard the absorption of the hemorrhage in the vitreous." (Lauber, *I. C.* p. 90) are suggestive of his assumption that the normal lymph stream and the disintegration of the blood are not alone sufficient for carrying out the absorption on a larger scale, but that the active co-operation of living cells is necessary.

In the following pages I want to show that the *non-pigmented cells of the epithelium*, especially *of the flat part of the ciliary body*, are able to absorb, to digest, and to carry away decomposed masses of blood; that, under certain conditions, a broad area can absorb blood from the vitreous, and occasionally may dispose of even large amounts of blood in a surprisingly short period of time.

Histological examination of eyes with hemorrhages has occasionally suggested these ideas, but the changes in the cells were always so insignificant that no positive statements

could be made regarding the activity of these cells in the absorption of vitreous hemorrhages.

In the case I am going to report, the phenomena in the cells were so marked that any doubt as to their nature and significance is excluded. I want to describe only the most significant details of this case.

The patient, male, aged 63 years, whose right eye was injured 17 days ago by the horn of a cow. A typical rupture of the sclera was present externally and superiorly, 1*cm* behind the limbus, with considerable suffusion of the conjunctiva. Anterior chamber normally deep, pupil round, iris brownish-red (the left eye was blue) with distinct structure, lens transparent. From the vitreous, sanguineous reflex. T. n. L. P. in 3*m;* projection only temporal. Enucleation. The eye, divided into horizontal series, shows an atypically situated sclerotic rupture, externally, a little in front of the equator. A piece of the retina healed into the cicatrized site of the rupture. Posteriorly the retina was detached by blood. In the anterior chamber was a hemorrhage consisting of red blood corpuscles in the form of tiny particles and here and there groups of granules containing blood-pigment. Surface of iris likewise covered by tiny particles. The protoplasm of the endothelial cells of the cornea was tinged reddish by blood-pigment. Tiny reddish granules were also perceptible in the cells. The upper surface of the anterior limiting layer of the iris represented a distinct brownish line. The lens was in the right place, the epithelial cells of the capsule of the lens in the region of the pupil, and above, behind the iris, were likewise tinged reddish.

The vitreous and the epithelial cells of the ciliary body showed the following points of interest:

The whole vitreous was infiltrated by blood. But during fixation the mass contracted in a posterior direction and a part of the mass dropped out during the preparation. The red blood corpuscles preserved their form only in a cup-shaped zone surrounding the posterior lenticular surface as well as in the inner and lower half of the eyeball, where a layer of several *mm* thickness extended from the region of the ciliary body to the vicinity of the equator. The blood corpuscles were transformed into a detritus of tiny granules, imbedded in a reddish, probably previously liquid, stroma. These granules occupied chiefly the posterior part of the hemorrhage, while the anterior sections of the vitreous body were practically free. There were practically no preserved

red blood corpuscles found next to the layers. Even in the vicinity of the ora serrata they were separated from them by a narrow space. This interspace contained blood corpuscles in an homogeneously tinged substance. Phagocytes containing disintegrated blood corpuscles were found between the fibers of the zonula, as shown in illustration 20, plate XI of Fuchs' article.

Quite a striking picture is offered by the cells of the inner non-pigmented layer of the flat portion of the ciliary body in its whole extent. While elsewhere the protoplasm of these cells presented a completely transparent appearance in eosinhemalum stained sections, here it had become opaque through the absorption of a foreign substance. In the sections, only slightly stained with eosin, this substance appeared yellow. In the preparations more deeply stained with eosin this substance had taken on a deep yellow-red color, which was even apparent with the low power. (See illustration.) In general, the bodies of the cells were permeated with a homogeneous mass which was also found upon the inner surface of the cells, whereby the cell outlines towards the vitreous became indistinct. The contents were not always homogeneous, light as well as deep red portions were distinguishable; and we encountered irregularly shaped, lumpy masses which in certain spots appeared as remnants of broken-down erythrocytes. Vacuoles, dispersed between them, had possibly proceeded from a contraction of these masses, or originated in the fixation fluid. The contents filled the whole cell up to the pigment layers, though they were present in greater masses in that part of the cell next to the vitreous. The nuclei of the cells were normal. From the pigment layer pigment granules had invaded the internal layer. Any doubt as to the content of the cells being altered red blood corpuscles, and detritus therefrom, would be dispelled by the fact that phagocytes lying in the vitreous and which have absorbed blood corpuscles, showed the same yellowish red crumbly contents. It has already been mentioned that only at some distance from the inner layers of the eye preserved red blood corpuscles were to be found, while in the peripheric parts of the vitreous the red blood corpuscles appeared as detritus and disintegrated granules. The contents frequently consisted of several confluent epithelial cells, so that the individual cell-bodies could not be distinguished and the cells were distended by their contents.

Groups of large cells with oval nuclei were found upon the epithelium. These were cell-nests which had their origin in the proliferation of the cells of the unpigmented

layer. They had the same contents, derived from the red blood corpuscles, as the epithelial cells.

This cellular "imbibition"—if we may use this term—ceased towards the folded portion of the ciliary body, and in the region of the processes the cells were free of these contents.

In the tissue of the anterior part of the retina, in the region of the ora serrata, this substance was present just as in the cells of the flat portion of the ciliary body.

It was difficult to decide on the participation of the retina in the absorption processes, though in various places Mueller's supporting fibers seemed to be saturated with the same substance which also extended into the interior of the retinal parenchyma. In the inner retinal layers large mononuclear phagocytes were found not infrequently filled with fine, granular detritus derived from disintegrated erythrocytes, though no hemorrhage had occurred in this part of the retina.

When a hemorrhage had taken place beneath the retina, this substance was absorbed by the layer of rods and cones, the ends of which, without distinct demarcation projected into the blood.

This substance could be traced as far as the membrana limitans externa and through its interstices into the external granular layer.

The substance contained in the epithelial cells of the ciliary body did not give a Berlin blue reaction, and only a few phagocytes were stained blue.

The perivascular lymph spaces of the retina showed no change except a slight increase of round cells.

As already mentioned, I have often observed in the cells of the flat portion of the ciliary body a substance derived from extravasated blood in the vitreous but these changes were never pronounced enough to allow a definite conclusion. For instance: Ten months ago an operation for cataract, with loss of vitreous, was performed on a man aged 63 years, with pronounced myopia. Vision at first was good, but a month ago a spontaneous hemorrhage occurred in the eye, which was followed by other hemorrhages. In sections stained with eosin-hemalum the protoplasm of the epithelial cells of the flat portion of the ciliary body was not clear and transparent, but of a dull yellowish-red colour, apparently from imbibition of a foreign substance. In my opinion absorption

of disintegrated blood by the cells of the flat portion of the ciliary body is of more frequent occurrence than one would think, judging from the fact that the activity of these cells has hitherto escaped notice. The changes in the cell protoplasm are as a rule so slight, the unusual contents are so inconspicuous, and hardly noticeable even with intensive staining of the protoplasm with eosin, and by their crumbly appearance, that an oversight is quite understandable.

I knew that exceptionally in eyes with vitreous hemorrhages erythrocytes can be absorbed unchanged or broken up into lumps by the pigmented cells of the ciliary body. As a case in point, I want to quote a traumatic hemophthalnus where, seven days after the injury, the eye was enucleated. The erythrocytes contained in the vitreous hemorrhage were unchanged, in many places the cells of the flat portion of the ciliary body had absorbed red blood corpuscles which, in form and appearance, showed hardly any change. Such an absorption probably takes place only under certain conditions.

The non-pigmented cells, while still in their normal relations, may develop phagocytic activities. Fuchs has called attention to the phagocytic activity of cells with retinal pigment. But under the influence of a hemorrhage such cells may become detached and mobile and emigrate into the blood mass. This detachment has not been observed in the case of the non-pigmented cells, though they are earlier and more intensively attacked by the stimulus. This phagocytic activity of the non-pigmented cells of the ciliary body, and its significance in the absorption of vitreous hemorrhages, is overshadowed by that activity which was so evident in the case reported above and which is interpreted as a manifestation of the normal biological activity of these cells. In addition to the function exercised by the cells of the ciliary body in its secretory activity, various clinical observations suggest that these cells exhibit also an absorptive activity, that they absorb fluid and therein contained substances from the vitreous, and pass them on. If, in the presence of iron salts in the vitreous, we find the cells of the ciliary body saturated with them, this phenomenon is not to be interpreted in the purely physical sense of diffusion, but rather that these cells in their biological function continually absorb fluid from the

vitreous and that when foreign substances are present, these also are incorporated into their protoplasm. Thus, these substances may accumulate in the cells, or they are carried on and can then be demonstrated in the lymphatic spaces of the retina.

In my essay on *Chronic Irido-cyclitis and Neuritis Retrobulbaris* (Fuchs' *Festschrift*), I suggested the possibility that retinal periphlebitis may also develop from the cells of the ciliary body absorbing toxins or bacteria and conducting them along the lymph circulation posteriorly towards the retina, where finally they enter the channels of the perivascular lymphatic vessels, to settle there.

Another question suggests itself, why does not this activity of the cells start in in every case of intraocular hemorrhage and lead to a rapid absorption?

This is due to several circumstances. For the absorption of larger amounts of blood in the vitreous the blood must undergo a certain transformation so as to render it suitable for absorption through the ciliary epithelial cells. Phagocytosis of unchanged, or only slightly changed erythrocytes need not be considered. The hemorrhage must be converted into a more or less homogeneous mass by the disintegration of its cells before it is suitable for absorption into the body of the epithelial cells. We do not yet know what is necessary to bring about this disintegration.

In his article (p. 222), Fuchs enlarges on the cause of hemolysis in the vitreous, without reaching a definite conclusion. There are several substances occurring in the vitreous which would have to be considered. But the vitreous does not contain a greater amount of inorganic salts, to which the hemolysis is due, than the blood. The same holds true of certain organic substances with hemolytic effects, such as urea and lactic acid. Furthermore, we know nothing regarding the physical and chemical nature of the diseased vitreous. We must also take into consideration that the hemolysis is the result of the death of the erythrocytes, caused by insufficient supply of oxygen, as Fuchs has shown that hemolysis occurs frequently, and preponderatingly, in the centre of large hemorrhages. As we frequently find erythrocytes of normal appearance intermingled with "blood-

shadows," individual chemical differences of the single erythrocytes would play a determining rôle according to Fuchs' view. But hemolysis is only one out of many factors which may effect a disintegration of the blood exudate.

It is probable that under certain conditions the ciliary epithelial cells exercise a cytolytic influence upon the red blood corpuscles, but how far this influence reaches we do not yet know, and this problem has not yet even been taken into consideration.

Prompted by Roemer's experiments—according to which blood could be rapidly removed from the vitreous of a rabbit eye if a few drops of immune serum from the guinea-pig, which was hemolytic for the rabbit blood, was injected in the vitreous, Elschnig (*A. f. A.* 51, Bd. p. 354. *Wiener Ophth. Ges.* 10, 2, 1904) tried to produce absorption of hemorrhages in the vitreous of the human eye. But the experiment miscarried because of a severe irido-cyclitis occurring after the injection of the hemolytic immune serum in the vitreous and necessitated enucleation.

In Elschnig's opinion, the hemolysin had not only destroyed the red blood corpuscles, but had severely injured the tissues of the retina and caused their necrosis.

Of course, we have to take into consideration whether, under the rapid disintegration of the red blood corpuscles, toxins might not have developed which in turn injured the tissues or that the tissues through previous hemorrhage react in an anaphylactic manner.

It is a well-known clinical phenomenon that repeated effusions of blood into the interior of the eye are followed by irido-cyclitis, and a slow irido-cyclitis occurring after intraocular hemorrhages (the same as in venous thrombosis) is a characteristic clinical picture.

It has not yet been explained, though it would be worth while investigating, to what extent diseases of the blood-vessels with involvement of the perivascular lymph sheaths cause disturbances in the outflow of the tissue fluid, which act as inflammatory irritants in these eyes.

And we are entirely in the dark regarding the extent to which the activity of the epithelial cells is increased by certain factors, so that they are capable of absorbing and

changing large amounts of the appropriately prepared blood. It seems quite probable that it is only after the establishment of both conditions that an active absorption of the blood by the epithelial cells takes place, and this may also be the reason that so little has so far been reported regarding these processes. Moreover, owing to the favorable clinical course, just such eyes have in most instances not been accessible to pathological investigation.

That, in our case, it was primarily a change in the blood which promoted absorption by the cells, is evidenced by the fact that we could show that the absorption of blood took place by the endothelium of the cornea and iris, as well as by the epithelial cells of the capsule of the lens—a process which is to be regarded as nothing other than the expression of a physiologic activity (absorption of intraocular fluid), which the histological investigation detected in the peculiar quality of the fluid.

Our observations show that, in the removal of hemorrhages in the vitreous body, the epithelial cells of the ciliary body—and possibly also the retina at the ora serrata—have a larger share than may appear from our present knowledge. We may hardly be wrong if, from all this, we draw the conclusion that these cells also represent an outlet for the intraocular fluids from the eye. It does not seem probable that the sole function of these cells is the absorption of morbid substances from the vitreous, but we are more justified in assuming that the regular function of these cells—as well as of the anterior part of the retina in the vicinity of the ora serrata—consists in the absorption of fluid from the vitreous and its transmission along the lymph paths. In places where these paths are lacking, the fluid accumulates within the tissues and expands by distending the tissue clefts (Blessig's cysts), while further back, where the blood vessels of the retina with their perivascular lymph sheathes are located, regular removal of the fluid is possible without infiltration of tissue.

This perhaps gives a better explanation for the so-called "vitreous glaucoma," which would occur if the vitreous loses the capability of discharging fluid, or if the cells of the ciliary body cannot absorb fluid from the vitreous. This would

also explain the futility of surgical interference in such cases of glaucoma.

The activity of these cells may also exercise a certain influence upon other morbid substances in the vitreous body and explain the surprisingly rapid absorption of vitreous opacities of inflammatory origin.

In his article on the absorption of vitreous exudates, Fuchs declares that, after its disintegration, the blood is discharged through the regular channels. This is in full agreement with our views, except that we have called attention to an additional channel, the significance of which had hitherto not been recognized.

# THE EXPERIMENTAL TRANSMISSION OF ARSENIC TO THE AQUEOUS HUMOR.[1]

By A. M. YUDKIN, A. C. KRAUSE and D. G. MORTON, New Haven

(From the Section of Ophthalmology, Department of Surgery, Yale University School of Medicine.)

## Introduction.

With the introduction of the arsenicals and bismuth compounds into the treatment of syphilis in general, great improvement has resulted in the treatment of syphilitic eye conditions. The intravenous use of the arsenicals combined with the older forms of therapy has materially shortened the time required to effect a cure, thereby reducing the number of more permanent injuries which follow long-standing ocular syphilis. In general, according to Rea (1) the time has been reduced from about twelve months to six by the use of the arsenicals.

The transmission of substances from the blood stream to the aqueous humor brings up a multitude of questions far too numerous to discuss here. Suffice it to say for the present that a considerable number of substances have been found in the aqueous humor after intravenous and subcutaneous injection; fluorescein, the iodides, mercury, and potassium ferrocyanide are some of the more prominent ones. There are various theories concerning the formation of the normal aqueous humor and the refill after paracentesis of the anterior chamber. The preponderance of evidence seems to be

[1] Preliminary report in the Proceedings of the Society for Experimental Biology and Medicine, 1927, xxiv, pp. 384–385.

in favor of the theory that the normal aqueous humor is formed by a process of filtration from the blood stream through the ciliary body, although this is by no means a settled question. It is known that the fluid which fills the anterior chamber after paracentesis is quite different from the normal aqueous humor, in that it contains a considerably larger quantity of protein and solids, and is coagulable.

Undoubtedly substances in the aqueous humor have a profound effect upon the eye, and especially the parts intimately associated with the anterior chamber; it is for this reason that the arsenic content of the aqueous humor after the intravenous injection of an arsenical is important. If we had a practical method of increasing the arsenical content of the aqueous humor it seems reasonable to suppose that this would be of great value in the treatment of ocular syphilis. It was with these points in mind that the following investigation was undertaken.

In a search through the literature only two references were found to work done on the arsenic content of the aqueous humor after the intravenous administration of an arsenical. In 1922, Neame and Bury (2) injected 0.3*gm* of novarsenobenzol into the ear veins of three rabbits. Two hours later the rabbits were killed. At ten minutes post mortem the aqueous humor was removed with a fine needle from five eyes. The fluid obtained was pooled, digested, and tested for arsenic by the Marsh method. No arsenic was detected. In 1923, Neame and Webster (3) made a further study of the situation, and on the basis of their finding suggested that paracentesis of the anterior chamber shortly after the intravenous injection of an arsenical might prove of value in the treatment of syphilitic interstitial keratitis.

## Procedure.

Four experiments were done, using eleven dogs in all. Small, young, healthy dogs with eyes of normal appearance were selected, to which no arsenic in any form had ever been given as far as could be determined. Fifty milligrams of neoarsphenamine per kilogram of body weight, dissolved in freshly distilled water, was injected intravenously (into the

jugular vein) in every instance. Aqueous humor was collected from both eyes at varying intervals of time after injection, as for instance, at 1, 2, 4, 8, and up to 96 hours. The fluid from the two eyes was pooled in all cases. As much fluid as possible was taken, but no attempt was made to measure the exact amount. Approximately 1.5*cc* was obtained at first tappings; second and third tappings, particularly when made at short intervals of time (few hours) after first tappings resulted in smaller amounts, approximately 0.85*cc*. The pooled fluid was then digested (4) with concentrated sulphuric and nitric acids for one hour, and longer whenever necessary, neutralized, and tested for arsenic by a modified Gutzeit method (5).

Attention must be called to the fact that these studies are in no sense strictly quantitative. This is not to say that the accuracy of the actual determinations was in any way slighted, but rather, that emphasis was put on the relative rather than the actual values. The method purports to detect arsine in amounts as low as 0.001*mgm;* the values obtained are of relative significance, and of course, qualitatively, positive results are positive and negative negative. No claim is made to actual values because the fluid obtained at second and third tappings, particularly when made at short intervals of time after the preceding tapping, was quite obviously different in character; the samples were smaller in amount, richer in protein and solids, and often had a web-like material forming in them. In order to measure such material accurately it would have to be weighed; in addition, the weights would be of no value unless simultaneous specific gravity determinations were done, a procedure entailing prohibitive difficulties. Therefore, since the relative values were considered of practical worth and could be readily obtained by using a standard amount of humor each time, this method was adopted, and the standard amount was set as all that could be obtained.

In two instances tappings were made before the injection of neoarsphenamine. These samples were run through in the usual manner, and served as controls, no arsenic being found in either case. In no instance in which the eyes had become congested, or in which the aqueous humor obviously contained blood was the fluid used. The eyes were anæs-

thetized with a few drops of a six per cent. solution of cocaine, a sharp fine bore needle pushed through the sclero-corneal junction into the anterior chamber, and the fluid slowly withdrawn into a *2cc* glass syringe, and subsequently discharged into a small Ehrlenmeyer flask. Needles and syringes were carefully cleaned before each operation.

The arsenic determinations were done by a modified Gutzeit method (5). Blanks were run on all the reagents, including the acids used in the digestion of the eye fluid, and found negative for arsenic. Briefly, the method depends upon the evolution of arsine by the action of hydrogen, liberated by zinc, on arsenious oxide, the reaction taking place in acid or alkaline solutions. The evolved arsine reacts with mercuric chloride, forming a colored compound. By the length and intensity of the stain the amount of arsenic is estimated by comparison with standard stains.

Results are expressed in micromilligrams of arsenic trioxide. One micromilligram of arsenic trioxide equals approximately 0.74*mmgm* of metallic arsenic. Traces were recorded when the stain was too short and too faint to be estimated quantitatively by comparison with the standard stains.

## Experimental.

In experiment 1, two dogs (1 and 2) were used; they were both treated in the same manner, each serving as a check on the other. One-half hour before injection of neoarsphenamine, paracentesis was performed. Tappings were then done after injection at the following periods, 1, 2, 4, 24 and 48 hours. No arsenic was found in the fluid obtained at either of the preliminary tappings. Comparatively large amounts were found at the one hour period, still larger amounts at the two hour period. From then on, the values gradually fell off to very small amounts at the 48 hour period. The results in the two dogs agreed remarkably well.

These results are interesting when compared with those obtained in experiment 2, which was carried out in exactly the same manner, using the same time intervals as in the first experiment, except that the preliminary tappings one-half hour before injection of neoarsphenamine were omitted.

In all except the one hour sample, the results tallied fairly closely with those obtained in the first experiment. However, in the one hour sample, only traces were found, whereas in experiment 1, comparatively large amounts were detected at this period,—*7mmgm* in one dog, and *8mmgm* in the other. The results of experiments 1 and 2 are recorded in Table 1.

TABLE 1.

| *Time Relation to Injection of Neoarsphenamine* | *Remarks* | Results | | | |
|---|---|---|---|---|---|
| | | *Dog 1* | *Dog 2* | *Dog 3* | *Dog 4* |
| 0.5 hour before..... | Aqueous withdrawn | 0 | 0 | | |
| | Injection | | | | |
| 1 hour after..... | Aqueous withdrawn | 8 | 7 | 0 | trace |
| 2 hours after..... | Aqueous withdrawn | 20 | 16 | 24 | 21 |
| 4 hours after..... | Aqueous withdrawn | 15 | 13 | 15 | 16 |
| 24 hours after..... | Aqueous withdrawn | 2 | 2 | 2 | 1 |
| 48 hours after..... | Aqueous withdrawn | 0.6 | 1 | — | 1 |

Results in micromilligrams of arsenic trioxide.

The next four dogs (5, 6, 7, and 8) may be grouped under one heading,—an experiment to find out whether or not the amount of arsenic in the untouched eye would increase as the time after injection of the arsenical increased. In the case of dog 5 the first tapping was done two hours after injection of neoarsphenamine; in the case of dog 6, four hours afterward; dog 7, twenty-four hours afterward; and dog 8, forty-eight hours afterward. Subsequent paracentesis was performed in all instances. The results tend to show that time has only a very slight effect. Traces were found in the first fluid of all these dogs except dogs 7 and 8 upon which first tappings were done twenty-four and forty-eight hours after injection respectively; in these two animals, *1mmgm* was found at the first withdrawal, such a slight increase over a trace that very little significance can be attached to it. The results of the subsequent tappings followed the general principle evolved in the first two experiments, namely, that larger amounts are found at the subsequent tappings, the values grading off as time goes on,—except that the amounts, as would be expected, were much smaller the longer after

injection the first tapping was made. The details and results of this experiment are recorded in Table 2. The findings in the case of dog 4 are included in the table for the sake of comparison.

TABLE 2.

| *Hours after Injection* | Results | | | | |
|---|---|---|---|---|---|
| | *Dog 4* | *Dog 5* | *Dog 6* | *Dog 7* | *Dog 8* |
| 1 | trace | | | | |
| 2 | 21 | trace | | | |
| 4 | 16 | 9 | trace | | |
| 8 | | | 2.5 | | |
| 24 | 1 | 1.5 | 1 | 1 | |
| 48 | 1 | 0.8 | trace | 2 | 1 |
| 96 | | | | 0.5 | 0 |

Results in micromilligrams of arsenic trioxide.

In experiments 1 and 2, it was definitely shown that if paracentesis be done one hour after injection, comparatively large amounts would be found at a subsequent tapping two hours afterward, and that fairly large amounts would be found at still another tapping two hours later. Experiment 4 was done in the hope of ascertaining whether once it had started to do so (initiated by a first tapping one hour after injection), and to find out how long the amount which did collect (as we have seen above, a large quantity could be counted on) would remain.

Three dogs (9, 10 and 11) were used. Each was given the standard injection. Eye fluid was removed one hour after injection in each instance. Traces were found in these samples, results consistent with those obtained in the first two experiments. On the first of these dogs subsequent paracentesis was performed at 4, 24, and 48 hours after injection; on the second, at 8, 24, and 48 hours after injection; on the third, at 24 and 48 hours. The details and results of this experiment, together with those for dog 4, which is included for the sake of comparison, are recorded in Table 3.

TABLE 3.

| *Hours after Injection* | Results | | | |
|---|---|---|---|---|
| | *Dog 4* | *Dog 9* | *Dog 10* | *Dog 11* |
| 1 | trace | trace | trace | trace |
| 2 | 21 | | | |
| 4 | 16 | 5 | | |
| 8 | — | — | 2 | |
| 24 | 1 | 1.5 | trace | trace |
| 48 | 1 | trace | trace | trace |

Results in micromilligrams of arsenic trioxide.

Apparently the greater part of the comparatively large quantity of arsenic which accumulates in the aqueous humor two hours after injection, following a tapping one hour afterward, soon disappears. The arsenic is either excreted, or the contents of the anterior chamber are largely replaced within the space of a few hours. After the first sudden decrease in the quantity of arsenic, that which remains seems to disappear much more slowly.

## Discussion.

The question of the transmission of substances from the blood stream into the aqueous humor is closely allied to the question of the transmission of substances from the blood into the cerebrospinal fluid. The exact mechanism of the formation of the aqueous humor and the spinal fluid is not known. The chief theories which vie with each other are the secretory and the filtration theories. Whichever is correct, the essential mechanism is probably identical in both cases. This similarity leads to some interesting comparisons which will be stated presently.

As mentioned in the introduction, a considerable number of substances have been injected into the blood stream and later detected in the aqueous humor. Memorsky (6) has shown that potassium ferrocyanide appeared in the aqueous humor 18 to 20 minutes after intravenous injection of 0.5*gm*

in dogs. Schläfke (7) demonstrated that potassium iodide appeared in the aqueous humor in 6 to 8 minutes after the oral administration of 0.25*gm* to rabbits. Hilbert (8) repeated Schläfke's experiment using the subcutaneous route, and found that the iodide appeared in the anterior chamber in 3 minutes. He also demonstrated that after the subcutaneous injection of iodoform in man, iodine appeared in the anterior chamber in 15 minutes. Similar experiments have been done by Leplat (9), Vinci (10), and Ottoglenghi (11). Traces of mercury have been found in the aqueous humor by Rampoldi (12) after subcutaneous injection of large doses of calomel. Ehrlich (13) employed fluorescein because of its coloring properties, and found it in the aqueous humor after intravenous administration, as a vertical green streak, the so-called Ehrlich's line. After eserin, Ulrich (14) found that the aqueous humor became more quickly colored with fluorescein. Wessely (15) obtained a similar result with pilocarpin. With the assistance of Hughson (16) in our laboratory, it has been shown that pilocarpin and eserine increases the arsenic content of the aqueous humor whereas atropine and cocaine have no effect on the transmission of this drug. The secretion of the aqueous humor has been artificially hastened in several ways; temporary luxation of the eyeball Nicati (17), Hamburger (18), cauterization of the limbus with silver nitrate, and the simple instillation of eserin or pilocarpin Wessely (19) have proved efficacious. Finally, all observers have noted that paracentesis of the anterior chamber, by decreasing the intraocular pressure and by producing a hyperæmia of the ciliary vessels, causes a rapid secretion of fluid to refill the chamber; a fluid which is coagulable, richer in protein and solids than the normal aqueous humor.

In like manner Kolls and Youmans (20) could find only traces of arsenic in the spinal fluid after intravenous salvarsan, even up to 72 and 96 hours later. In 1925 Fordyce, Rosen and Meyers (21) found comparatively large amounts of arsenic in the spinal fluid after the intravenous injection of neoarsphenamine in humans,—up to 48 hours after injection, the largest amounts being found at the 4, 5, and 24 hour periods. These observers performed only one puncture on a subject. Barbat (22) injected an arsenical into twenty-

six paretics and tabetics, performed a lumber puncture twenty minutes later, and again twenty-four hours later. In twenty-five of the fluids obtained on second puncture arsenic was found. In a considerable number of these fluids the amount was large. He attributed this phenomenon to an increase in the permeability of the meninges due to the decreased intraspinal pressure resulting from the first puncture.

This is striking when compared with the results obtained in this investigation, in which several paracenteses were performed shortly after the first one, which was done one hour after injection of the arsenical. Comparatively large amounts of arsenic were found in these subsequent specimens, whereas only traces were obtained in the first specimens. It is probable that the decrease in intraocular pressure and an increased hyperæmia of the ciliary body consequent upon a first tapping has much to do with this increase.

Of course, the amount of arsenic which can penetrate to the anterior chamber depends to a large extent upon the amount of arsenic available in the blood stream. Estimations for arsenic have been done on the blood after intravenous salvarsan at varying intervals of time after injection by Kolls and Youmans (20) in 1923. They found that approximately three-fourths of the arsenic injected had left the circulation within a few moments, but that the remaining arsenic disappeared much more slowly, days being required before it was all gone. In 1925, Fordyce, Rosen and Meyers (23) studied the arsenic content of the blood after the intravenous injection of neoarsphenamine. They found that the major portion disappeared almost immediately. They plotted the amounts against time and obtained a curve which drops rapidly during the first five minutes and then continues to decline much more gradually. These observers (24) also studied the partition of arsenic in the serum and clot after intravenous administration of the more commonly used arsenicals. In all instances they found higher values for arsenic in the serum at short periods of time after injection. Moreover, it was observed that the highest values for arsenic in the serum were obtained in the case of neoarsphenamine. A further study (25) of the partition of the arsenic between the blood stream and tissue, employing the usual arsenicals,

revealed that neoarsphenamine retained the highest percentage of arsenic in the blood stream (62%). Neosalvarsan was thus recommended as being the best drug for use in the Swift-Ellis treatment. We may safely assume that it was also the best of the arsenicals for the purposes of this investigation.

This investigation has been definitely demonstrated that paracentesis of the anterior chamber shortly after the intravenous administration of an arsenical causes an increase in permeability to arsenic. It has also been shown that the arsenic is quite rapidly excreted. Since paracentesis of the human anterior chamber is a fairly simple procedure, it would seem that its performance shortly after the intravenous injection of an arsenical is worthy of trial as a means of increasing the amount of arsenic which comes into contact with the syphilitic lesions. The possibility of arsenic poisoning of the structure of the eye must be borne in mind, although, according to Young and Loevenhart (26), there is little danger from the arsenicals except those which have the $NH^2$ group in the *p*-position. Neither arsphenamine nor neoarsphenamine come under this heading. Conceivably anything which artificially hastens the secretion of the aqueous humor would very probably increase the amount of arsenic penetrating to the anterior chamber after the injection of an arsenical. From the preliminary work in our laboratory we (16) have shown that pilocarpin and eserin increase the arsenic content in the aqueous humor. With this in mind, it appears that these miotics are valuable adjuncts in the treatment of syphilitic lesions of the ocular tissue particularly when it is desirable that the drug be introduced into the intraocular fluid.

## Conclusions.

1. After the intravenous injection of large doses of neoarsphenamine into dogs, only the minutest amount of arsenic, and sometimes none at all penetrates to the aqueous humor.

2. Paracentesis of the anterior chamber shortly before or after the intravenous injection of neoarsphenamine greatly

increases the permeability of the anterior chamber to the arsenic of the arsenical. This heightened permeability can be maintained by further paracentesis. This phenomenon is probably explicable on the grounds of the decreased intraocular pressure and the increased hyperæmia of the ciliary body caused by paracentesis.

3. The relatively large amount of arsenic which accumulates in the aqueous humor because of paracentesis near the time of injection of an arsenical leaves the anterior chamber in great part within a few hours.

4. Paracentesis of the anterior chamber, or some other means of increasing the rate of secretion of the aqueous humor near the time of injection of an arsenical, might prove of definite clinical value in the treatment of syphilitic affections of the eye.

5. The introduction of pilocarpin and eserin into the cul-de-sac also increases the arsenic content of the aqueous humor.

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# THE VALUE OF SCOTOMETRY IN THE DIAGNOSIS AND TREATMENT OF GLAUCOMA.[1]

By Dr. A. H. THOMASSON, New York.

SCOTOMETRY is probably the most important aid to the early diagnosis of glaucoma at our command. It enables us to discover an almost infallible sign of this disease long before any subjective symptoms are constantly present. The importance of an early diagnosis becomes manifest when we consider that unless such a diagnosis is made irreparable damage will be done, the tension will be controlled with much greater difficulty, and almost certainly an operation must be resorted to eventually to preserve whatever vision may remain after repeated attacks of high tension. Unfortunately, however, an early diagnosis is difficult if we rely upon the symptoms on which a diagnosis is usually based, for many of these symptoms are not constantly present during this early stage.

The symptoms of glaucoma are dependent, with possibly one exception (the adrenalin pupillary reaction), upon increased intraocular pressure, and late in the disease they are constant because they are then dependent upon anatomic changes in the eye that have been produced by the increased intraocular tension, and because at this stage of the disease the tension is usually constantly elevated. Early in the disease, however, before such changes as cupping, demonstrable optic atrophy, atrophy of the iris, shallow anterior chamber, etc., have taken place, the ordinary symptoms such as ven-

[1] Read before the Section on Ophthalmology of the N. Y. Medical Society at Niagara Falls, May 11, 1927.

ous congestion, steaminess of the cornea, halos, foggy vision, contraction of the visual field, etc., will only be present during a rise of the intraocular pressure. But during this stage of the disease the tension is not constantly above the normal. In fact, at this time there are frequent, and often fairly long periods, during which the tension is perfectly normal, and during which also there are no subjective symptoms. But a diagnosis must be made during this period in every possible case in order to prevent pathological changes that are so destructive to sight and that are responsible for practically all the complications that we encounter in our management of this disease. To defer a diagnosis in a case of glaucoma until obvious structural changes are present would be as inexcusable as it would be in the case of pulmonary tuberculosis to wait for obvious cavity formation and for the appearance of tubercle bacilli in the sputum before making a diagnosis. Each disease must be diagnosed in its incipient stage if we wish to prevent irreparable damage, and thus render to our patients the service they have a right to expect from us.

The great difficulty in the diagnosis of incipient glaucoma is that the patient may, and often does, present himself for examination during a quiescent period, when practically no symptoms are present. Such a patient will offer little in the way of history that is of value, and if our examination reveals an eye that is apparently normal we are apt to pass the case by without resorting to the tests that would enable us to make a diagnosis. Let us assume that such a patient comes to us for an examination. As has been stated, the history is usually unimportant. He is convinced that his sight is getting worse, and has made repeated attempts to improve it by a change of glasses. If closely quizzed concerning foggy vision, a certain percentage will recall such attacks, but the majority will not have noticed even this symptom. We proceed with our examination and find that the corrected central vision is normal. The field of vision, taken with a standard perimeter at 33*cm*, and using a 5*mm* object, is normal. We now examine the blind spots, using one of the short radius campimeters and a somewhat smaller test object, but by this test they are normal. The tension is normal. The pupils are of normal size and react to light.

This reaction may be somewhat sluggish, but this defect is easily overlooked in an otherwise apparently normal eye. There is no cupping that can be determined by the average examiner, and no optic atrophy can be made out. Have we not then done all that can be reasonably expected of us, and are we not justified in quieting our patient's fears and assuring him that his eyes are normal? Assuredly not, for we have neglected the two important tests that would have enabled us to make at least a tentative diagnosis of glaucoma, and we could then have placed the patient under observation until a definite diagnosis could be established. These two tests are: The examination of the blind spot for Bjerrum's scotoma, and the determination of the pupillary reaction to adrenalin. The second of these I shall not discuss in this paper, but Bjerrum's sign, although not absolutely pathognomic of glaucoma, yet,—because it appears at the very onset of the disease, does not disappear during periods of normal tension, is so constantly present when glaucoma is present and absent in non-glaucomatous conditions,—is the one outstanding sign of value during the early stages of this disease. I have never seen it absent in any early case of glaucoma, and have seen but one enlarged blind spot in a non-glaucomatous case that could have been mistaken for Bjerrum's sign, and this case did not bear the slightest resemblance to glaucoma in any other respect. The examination, then, of every suspected case of glaucoma must include a search for this sign, for when present it enables us to confirm a diagnosis of glaucoma in the absence of other definite symptoms, and when absent, it enables us to rule out such a diagnosis even in the presence of certain more or less definite symptoms, such as a large and unusual appearing physiological cup in a hysterical patient with a contracted field.

But Bjerrum's sign, when it first appears, can only be detected by Bjerrum's original method of scotometry, by means of a tangent screen, or some substitute for a tangent screen, at a distance of one meter or more, and by using test objects measuring from $\frac{1}{16}°$ to $\frac{1}{8}°$. That is, a *1mm* or a *2mm* object, if used at a distance of one meter. This fact has been repeatedly demonstrated by me, and in this paper the term "scotometry" has reference to Bjerrum's method of scoto-

metry. The reasons for using small test objects at a distance of at least one meter have been fully set forth in a previous paper.[1] This method of examination, then, becomes a necessary procedure in every attempt at the early diagnosis of glaucoma. When Bjerrum's sign has once been demonstrated, we should seek a confirmation of our diagnosis by determining the adrenalin pupillary reaction and by frequent testing of the intraocular tension, as well as by repeated examinations of the blind spot to determine whether there is any advance in the scotoma. By following some such procedure as this many cases of early glaucoma will be diagnosed that are now being overlooked, and when once the importance of scotometry has been learned, it will never be abandoned.

But scotometry is not only of value in the diagnosis of glaucoma. It is equally valuable as a means by which to determine the future progress of this disease, and as a guide to treatment. And this is true not only during the early stage, but throughout the entire course of the disease. When once Bjerrum's scotoma has been accurately outlined, if the treatment that is instituted, whether operative or miotic, is sufficient to keep the tension within normal limits at all times, there will be no change in the scotoma due to an advancement in the glaucomatous process. If a normal tension has been gained by means of an operation, the scotoma will usually not advance, but when a normal tension is apparently maintained by means of drops, there will usually be a steady advance in the scotoma. This seems to indicate that we cannot be sure of maintaining a normal tension by means of drops. In cases treated by this method there must, at times, be elevations of the tension of which we are not aware. But no matter what the method of treatment, we must keep ourselves informed at all times as to the progress of the disease. For this information we cannot rely entirely upon frequent determinations of the tension, for there are fluctuations of tension that occur in the interim between office visits, concerning which we can have no knowledge, and which steadily advance the glaucomatous process in spite of an apparently

[1] "A Simplified Tangent Screen with Suggestions on Field Taking." A. H. Thomasson, ARCHIVES OF OPHTHAL., vol. lv., No. 6, 1926.

normal tension. The same may be said concerning field taking by the ordinary method. The usual instruments for field taking do not reveal slight gains and losses in the visual field,—gains and losses that mean much in determining the dosage if a patient is to be kept on drops, also whether an operation is to be resorted to, and if so, when to operate. But scotometry gives us this information more quickly and more accurately than any other method of examination. It is, therefore, supplemented of course by other methods of diagnosis, the most reliable guide that we have as to the progress of this disease, and in the determination of the treatment. For this reason the scotometer becomes a necessary part of our office equipment if we treat glaucoma at all, for the physician who does not use it cannot obtain all the information concerning his patient that he should possess, and therefore places both himself and his patient at a disadvantage.

# THE NON-OPERATIVE TREATMENT OF CHRONIC GLAUCOMA.[1]

By Dr. SEARLE B. MARLOW, Syracuse.

SINCE Posey's paper in 1914 on "The Treatment of Chronic Simple Glaucoma with Miotics," very little has appeared in the literature about the non-surgical treatment of chronic glaucoma until 1923 when Hamburger reported his experiences first with adrenalin and later with glaucosan. The increasing interest in this method of treatment is evidenced by the fact that in the *Ophthalmic Year Book* for 1923 no paper dealing with medicinal therapy is listed whereas in the volume for 1926, 15 papers are recorded. The surgical treatment, on the other hand, has received and is constantly obtaining much attention. New operations are being proposed; old ones revived. Probably no one operation will ever meet the requirements of all surgeons.

The great preponderance of opinion as expressed in the majority of text books and in the discussion of papers relative to the treatment of glaucoma is that some operative procedure will sooner or later become necessary. The earlier in the course of the disease operation is done the greater the likelihood that the results will be beneficial and lasting. At the present time our knowledge of glaucoma and the various operations proposed for its cure does not permit us to say with certainty that operation will cure the disease. Moreover operative treatment can never be made entirely free from risk. The possibility of infection, both early and late, will always be present to a certain degree. Loss of vision

[1] Read at Annual Meeting, N. Y. State Med. Soc., Niagara Falls, May 11, 1927.

not infrequently results from a marked increase in astigmatism, hemorrhage and the rapid encroachment of a scotoma over the fixation point. Weeks believes that this will occur whatever the operation and perhaps in spite of any treatment. These facts are too well known to need extended discussion.

They serve, however, to emphasize the uncertainty of operative treatment. The proposal of new operations and the reviving of old indicate further not only the dissatisfaction with the available operations but also the failure of medicinal therapy. If non-operative treatment can be shown to be at least as effective as operative its advantages immediately become apparent. A large majority of ophthalmologists have probably observed one or more cases of chronic glaucoma controlled for a long time by medicinal treatment. These are selected cases and as a rule comprise solely those observed outside hospitals and clinics. Zentmayer suggests that miotics may be tried in patients where the tension does not range over thirty and in patients over sixty years of age. Calhoun advocates miotics in three groups of cases of simple glaucoma, (1) patients over 60 years whose life expectancy is not more than 10 years, (2) patients blind in one eye, (3) in those who have lost one eye as the result of operation.

The importance of thoroughly investigating the patient has long been recognized as essential not only for the purpose of diagnosis but also for treatment. In the discussion of a paper by Rowan in 1920 one speaker suggests that whether glaucoma is treated with drops or operation only a symptom is being treated and that if ever a cure is discovered it will be discovered by an internist. Imre's cases treated with glandular extracts and the cases of Lagrange and others associated with endocrine disorders lend strong support to this contention and indicate new possibilities. They serve also to emphasize the perhaps too frequently lost-sight of fact that it is the patient who needs thorough investigation rather than the glaucoma. In this connection the following case may be of interest.

Mrs. J. H. N., age 40, was referred by Dr. Edward Dennis of Erie to whom she had gone because of headache and slight blurring of vision. In the course of his examination he discovered the tension to be elevated, R. 35, L. 58,

Gradle Schiötz. Pilocarpin had controlled the tension so that when she was first seen two months later it was R. $18(\frac{5}{5\cdot 5})$, L. $29(\frac{2}{5\cdot 5})$ Schiotz. At this time vision of each eye was $\frac{6}{6}$ with her glasses. Both a.c. were deep, pupils 2–3*mm*, active, shallow cupping of both discs. The peripheral field of the R. eye was normal, the left somewhat contracted in the lower nasal quadrant, both blind spots enlarged for $\frac{3}{1000}$ test but no other changes. The treatment which Dr. Dennis had ordered was continued. Seven months later when she returned because of blurring the tension was R. $18(\frac{5}{5\cdot 5})$ L. $16(\frac{5\cdot 5}{5\cdot 5})$. Her glasses were modified with relief. After another two months her symptoms recurred but vision remained $\frac{6}{6}$. At this time the tension of the left eye was $36(\frac{3}{7\cdot 5})$, the right unchanged. Eserin, pilocarpin and cocaine were ordered to be used more frequently in the left eye. She was asked to see an internist at this time. Dr. W. A. Groat summarized his report as follows: "My impression was that there was endocrine imbalance, and I linked the inability to nurse her children, the progressive gain in weight with each pregnancy and the type of build as showing some pituitary dysfunction with possibly compensatory hyperthyroidism." In addition he advised X-ray examination of her teeth and the wearing of a truss for an umbilical hernia. In the meantime the tension came down to normal and she was not seen again for another eleven months. During the interval eleven abscessed teeth were extracted and she had worn the truss. Although no drops had been used for more than six months she had been entirely free from symptoms, had seen no halos the significance of which she understood thoroughly, and which had been regularly present prior to each previous visit. At this last last examination the vision of each eye was $\frac{6}{6}$, tension each $18(\frac{5}{5\cdot 5})$. She attributed her good condition to the extraction of the teeth and the wearing of the truss.

Until Hamburger's report in 1923 eserin and pilocarpin have been the chief remedies available for the medicinal treatment of chronic glaucoma. The use of adrenalin and glaucosan appear to be effective means of control in selected cases, some writers going so far as to state that by them operation can frequently be avoided. However, insufficient time has elapsed and their use by a large enough number of widely distributed observers is still lacking to permit the estimation of its real value. Moreover, the few unfavorable

reports in which elevation of tension had followed its use indicate that the cases must be carefully selected. Where this is done its trial seems more than justified. No cases treated in this way are available for this discussion.

The purpose of this paper is to report the results of miotic treatment in a series of 35 cases of chronic glaucoma which have been observed in the practice of my father, Dr. F. W. Marlow. The longest period of observation was 14 years in once case; the shortest 1 year; two-thirds have been observed for four years or less. The following table indicates the distribution.

| *Years* | *No. of Cases* |
|---|---|
| 14 | 1 |
| 10 | 1 |
| 9 | 1 |
| 8 | 1 |
| 6 | 3 |
| 5 | 3 |
| 4 | 3 |
| 3 | 5 |
| 2 | 10 |
| 1 | 7 |

In all but seven of these cases the miotics have been prescribed according to the formula of Wickerkiewicz which combines eserin, pilocarpin and cocaine in the following proportions:

| | |
|---|---|
| Eserin sulphat | 0.02 |
| Pilocarpin hydrochlorid | 0.40 |
| Cocaine hydrochlorid | 0.20 |
| Aq. dest. | 20.00 |

This prescription has as a rule been used only twice daily. In no case have iris adhesions become apparent and cases of toxic conjunctivitis have been exceptional. The impression gained from the extensive use of this combination is that it is more efficient and less annoying to the patient than either eserin or pilocarpin alone.

In order to discuss intelligently the treatment of chronic

glaucoma the two forms in which this condition occurs must first be recognized. The distinction between chronic inflammatory glaucoma and simple glaucoma is so well known that no elaboration is necessary. It does seem worth while, however, to restate Schweigger's view that chronic inflammatory glaucoma is dormant potential acute inflammatory glaucoma. It is for this reason that operation is almost universally considered to be necessary early in this type of the disease.

| *No. of Cases* | *Age at Onset* | *Average Duration* | Chronic Inflammatory | | Simple | | *Operated* |
|---|---|---|---|---|---|---|---|
| | | | *Total No.* | *One Eye Blind* | *Total No.* | *One Eye Blind* | |
| 4 | 30–39 | 6 yrs. | 2 | 1 | 2 | 0 | 1 (S) |
| 9 | 40–49 | 10.5 " | 3 | 1 | 6 | 4 | 2 (CI) |
| 9 | 50–59 | 8.3 " | 3 | 0 | 6 | 1 | 1 (S) 1 (CI) 1 (CI) |
| 9 | 60–69 | 3.8 " | 3 | 1 | 6 | 2 | 1 (CI) |
| 4 | 70–80 | 2.5 " | 0 | | 4 | 0 | 0 |

This table shows that in nearly two-thirds the age at onset was 50 or more. It was under 50 in 5 cases of the chronic inflammatory type, over 50 in 6; of the simple case 8 were under 50, 16 over 50. This suggests a more even distribution for the chronic inflammatory cases and a great preponderance of the simple at a greater age. Ten patients were blind in one eye when first seen. Six of these were under 50 at the age of onset, 4 over. Two of three chronic inflammatory, our of seven simple cases which were blind in one eye were under 50 at the onset of the disease. Seven cases came to operation, three being under 50 at the age of onset. These facts seem to indicate that no matter what the type of the disease the younger the patient the more devastating its effects. Although the teaching that conservative treatment should be used only in cases over 60 receives some support,

its use in much younger patients appears beneficial for long periods of time in both types.

Eleven cases of chronic inflammatory glaucoma are included. Four were in an advanced stage of the disease when first seen. They are the first, fourth, fifth and tenth cases in the list. Case No. 5 was the only one favorably influenced by miotic treatment. This patient was also the only one under fifty at the age of onset and had been observed for five years at the end of which time vision was better than at the original examination. The first case was seen only at long intervals and very probably had not followed out the treatment consistently. One patient refused operation, miotics maintaining vision for one year. One patient was successfully operated so far as tension was concerned but with no influence upon vision. Thus miotic treatment was advantageous in one case only.

The seven early cases had their age at onset of 35, 42, 36, 50, 52, 64 and 45 years. Four subsequently came to operation. One was operated elsewhere in spite of the fact that miotics had improved and maintained the vision and tension for more than 1 year. One developed an acute attack after 6 years necessitating iridectomy with good results. In one iridectomy failed to check the loss of vision. In the three cases not operated the vision was maintained for 14, 2 and 1 years. From this the conclusion may be drawn that the advanced cases of chronic inflammatory glaucoma in this series progressed unfavorably in the majority of cases in spite of treatment, operative or otherwise. In the early cases operation benefited 2, and had no effect in one, the effect of operation in one being unknown. On the other hand three cases were favorably influenced, one for a considerable period of time. Therefore, it seems justified to state that miotics, in some cases at least, are capable of controlling the progress of the disease and that their use for a long time does not necessarily preclude good results from eventual operation. It also appears true that the younger the patient the more likely is operation to become necessary.

Twenty-four cases of simple glaucoma are summarized as follows. Ten were in an advanced stage of the disease when they were first seen. In one case iridectomy was done on one

eye with apparent arrest of progressive change, the unoperated eye remaining under miotic treatment. In two consultants advised against operation. Miotics maintained or improved vision in five cases including one in which operation was considered inadvisable. Five cases failed in spite of treatment. The more rapid failure took place in the older patients so that one patient had only P.L. after 4 years, one $\frac{6}{60}$ after 2 years, and one went down from $\frac{6}{12}$ in each eye to R.$\frac{6}{18}$, L. $\frac{6}{24}$ in one year. The first two patients were 65 when first seen, the last 75. The failure of the two younger patients, one 49, the other 37 at the first examination, was from $\frac{6}{6}$ to $\frac{6}{12}$ in 4 years and from $\frac{6}{6}$ to $\frac{6}{18}$ in 3 years. The rate of change was slow in each case, one of which was operated. These cases seem to indicate that older patients with advanced disease respond less readily to miotics than younger. Miotics are, however, advantageous in advanced cases, only two cases of this series becoming seriously handicapped while under observation. Of fourteen early cases the vision of five at the last examination was less than at the first. One case was operated after nine years of observation. This was a case of high myopia in which vision continued to fail and the field to contract in spite of the fact that miotics kept the tension at a normal level. A Lagrange operation was successful in bringing the tension to a normal level but failed to check the advancement of a scotoma and further loss of vision. In two the visual loss was accounted for by the development of lens changes. In another advanced cardio-renal disease with retinal changes was an unfavorable underlying factor. In the last the vision had fallen from $\frac{6}{6}$ to $\frac{6}{9}$ after 6 years due to the glaucoma although the tension was not greatly elevated. In this case a consultant advised operation. The remaining nine cases maintained the vision of the first examination so that miotics apparently checked the progress of the disease.

Thus with two exceptions all the early cases did well no matter what the age at onset. While operation may eventually become necessary in some of the younger patients this is less likely to occur in older patients. The teaching that patients of 60 years or over can be safely treated with miotics gains some support from these cases. They further suggest

that the value of this treatment is not limited to patients of this age and that not a few cases can be carried along safely for a considerable period if they are carefully watched.

In 1901 DeSchweinitz, in a paper read before the American Ophthalmological Society, advocated iridectomy on the second eye at the earliest possible moment in all cases of acute and chronic inflammatory, and simple glaucoma if any signs of the disease could be demonstrated. The general trend of the discussion supported his opinion. In 1920 in the discussion of the paper of Rowan, already referred to, a less radical attitude was manifest although some further support of this opinion is offered. It is interesting in this connection to note the results of non-operative treatment in the patients in this series who were blind in one eye when first examined.

Ten such cases are listed as follows:

| | *Age at 1st Exam.* | *Period Observed* | *Vis. at 1st Exam.* | *Vis. at Last Exam.* |
|---|---|---|---|---|
| Chronic Inflammatory.... | 68 | 2 yrs. | L $\frac{6}{\frac{1}{6}}$ | H.M.* |
| | 45 | 6 | L $\frac{6}{6}$ | $\frac{6}{6}$† |
| | 37 | 15 | L $\frac{6}{6}$ | $\frac{6}{6}$ |
| Simple..... | 73 | 4 | L $\frac{6}{\frac{1}{60}}$ | $\frac{6}{6}$ |
| | 65 | 2 | L $\frac{6}{6}$ | $\frac{6}{6}$ |
| | 64 | 3 | L $\frac{6}{6}$ | $\frac{6}{6}$ |
| | 58 | $1\frac{1}{2}$ | R $\frac{6}{6}$ | $\frac{6}{6}$ |
| | 52 | $3\frac{1}{2}$ | L $\frac{6}{6}$ | $\frac{6}{6}$ |
| | 51 | 1 | L $\frac{6}{\frac{1}{15}}$ | $\frac{6}{\frac{1}{15}}$ |
| | 49 | 4 | R $\frac{6}{6}$ | $\frac{6}{\frac{1}{12}}$ |

* Operation refused. † Iridectomy for acute attack $\frac{6}{\frac{1}{12}}$.

Two cases failed rather rapidly and one developed an acute attack. In the case that failed from $\frac{6}{6}$ to $\frac{6}{\frac{1}{12}}$ after four years a consultant advised against operation. The remaining cases had as good or better vision at the last examination than at the first. They would seem to support Calhoun's suggestion that patients with one blind eye should be treated with miotics. The fact that one eye is blind may be contributory to the good results obtained for two reasons. First being

blind in one eye the diagnosis is made earlier in the second. Second, having but one eye the patient is correspondingly concerned and takes better care of this eye.

Although the treatment appears to have maintained the vision in the majority of these cases during the period of observation it has been less effective in arresting the contraction of the field of vision. In only one case was the field of vision larger at the last examination than at the first. In many of the early cases the progressive contraction of the field appeared slowed. In two cases which had normal periphery but typical scotomata the scotomata remained stationary under observation. In one case observed for nine years before operation while the tension and central vision were maintained the field progressively changed. Operation in this case while successful in reducing the tension did not check the advance of the field changes. In the advanced cases treatment seemed to have much less effect.

The intra-ocular tension was kept within normal limits in 8 chronic inflammatory cases and 16 simple cases. The three remaining chronic inflammatory cases were all operated on with a good result. Two others were operated without checking the progress of the disease. One of these was in an advanced stage, the other in an early stage of the disease. In four of eight cases of simple glaucoma in which the tension was not controlled by miotics the vision improved in one eye. One maintained the vision of the first examination for over a year although the tension was considerably above the normal. Two failed and may have been operated elsewhere. One had rather marked arterial disease. All but one were advanced cases. The one early case was the case with arterial disease. This constitutes on the whole a favorable showing for the treatment employed. It further indicates that increased tension is only a part of the disease process.

Although this series of cases is too small to allow the drawing of general conclusions, they seem to suggest that non-surgical treatment is justified in a large number of cases, They also emphasize, what is already generally accepted. that treatment, surgical or medical, is more likely to be successful when supplemented by careful and thorough investigation of the patient.

CASES OF CHRONIC INFLAMMATORY GLAUCOMA.

1. Mrs. P. (18–186) 60 years. 1892. Myopic. Symptoms for 7 years. Each $\frac{6}{24}$. T.R.–N. L + 2. Cupping, nasal loss of field. Eserin. Not seen subsequently until 1906 (14 years). Blind.

2. Mrs. E. T. (33–52) 35 years. 1896. Hyperopic astigmatism. Anterior chamber shallow. Pupils oval, sluggish. T + 2 each. Deep cupping. Vision $\frac{6}{12}$ each. Nasal loss of field. Eserin, later pilocarpin. Last seen 1897 (1¼ years). R. $\frac{6}{9}$., L. $\frac{6}{9}$.

3. Mrs. J. R. (55–102) 36 years. 1902. Compound hyperopic astigmatism. Glaucoma 1911. Symptoms 2 years. R. blind, L. $\frac{6}{9}$. T. R. 60 L. 34. Shallow a. c. large pupils, deep cupping. Field L. normal. E. P. C. 1917, acute attack. L. Iridectomy. 1927. $\frac{6}{12}$. T. 25. Typical field loss.

4. Mrs. P. (67–185) 68 years. 1905. R. Blind. L. $\frac{6}{24}$. Tension + 2. Pupils dilated. Operation refused. E. P. C. Vision held 1 year. 1907 L. Hand movements in outer part of field.

5. Mrs. M. J. H. (82–174) 46 years. 1909. R. $\frac{6}{18}$, L. $\frac{6}{24}$. Shallow a.c. Pupils dilated. Arterial pulsation. T. R. + 2. L.+?. E.P.C. 1915. R. $\frac{6}{12}$., L. $\frac{6}{9}$. T. 25–30 each.

6. Mrs. W. G. S. (88) 37 years. 1910. Symptoms 1 year. R. Blind, L. $\frac{6}{9}$. T.R. + 3 L.N. to fingers. Glaucoma L. 1920. $\frac{6}{9}$. T. 70. Shallow a.c. Dilated immobile pupils. Nasal loss of field. E.P.C. Last seen 1927 $\frac{6}{9}$. T. 22.

7. Mrs. L. E. G. (1048) 47 years. 1911. Pterygium. Glaucoma 1915. R. $\frac{6}{9}$, L. $\frac{6}{18}$. T. R. 30 L. 60. Cupping. Nasal loss. E.P.C. 1917 Iridectomy L. continued failure. After 8 years. R. $\frac{6}{9}$ T. 18.

8. Mr. A. W. D. (3655) 56 years. 1915. Symptoms 4 years. R. $\frac{6}{12}$, L. $\frac{6}{9}$. T. R. 55 L. 40. Pupils large, inactive, shallow a.c. Nasal loss of field E.P.C. 2 years. R. $\frac{6}{12}$, L. $\frac{6}{9}$. T. R. 31 L. 22.

9. Miss A. E. (5096) 66 years. 1916. Iridectomy L. 2 years, before, R. $\frac{6}{9}$, L. $\frac{6}{18}$. T. R. 31 L. 36. Hyperopic. Shallow a.c. Cupping. E.P.C. After 1 year. R. $\frac{6}{9}$ T. 26.

10. Mr. E. L. G. (4428) 67 years. 1916. Emmetropic. R. $\frac{6}{18}$, L. $\frac{6}{24}$, T. R. 37, L. 40. Pupils large inactive. Field N. Cupping. E.P.C. After 2 years. V. $\frac{6}{24}$. T. 35 each. Iridectomy lowered tension but vision continued to fail.

11. Mrs. M. K. (6526) 48 years. 1918. Hyperopic. Symptoms 3 years. Pupils large, sluggish. No cupping. V. $\frac{6}{9}$? T. R. 70, L. 43. E.P.C. 1920 V. $\frac{6}{18}$ each, T.R. 50 L. 28. Iridectomy R. Iridotasis L. 1922 V. R. $\frac{6}{24}$ L. $\frac{6}{18}$, T. R. 18 L. 8.

### Cases of Simple Glaucoma.

1. Mr. S. (1–130) 73 years. 1885. R. Eye Blind for 6 years. L. Eye $\frac{6}{18}$. T. + 2, deep cupping. Nasal loss of field. Myopia. Eserin. Three years later $\frac{6}{18}$.

2. Miss H. (5–87) 31 years. 1887. High myopia. Vision $\frac{6}{9}$ each with correction. Glaucoma 1906. R. $\frac{6}{12}$, L. $\frac{6}{9}-$. T. N. to fingers. Nasal loss of field. Deep cupping. E.P.C. Maintained tension. Progressive loss of field. Lagrange operation 1916. Tension controlled. Field progressively diminished. Last seen 1927. L. $\frac{6}{60}$. Central scotoma. Tension N.

3. Mr. S. (31–61) 39 years. 1895. Compound hyperopic astigmatism. Glaucoma 1924, age 68, $\frac{6}{9}$ each. T. R. 26, L. 18 (Schiotz) Typical scotoma R., enlarged blind spot L. E.P.C. Observed 2 years. Tension 18 each. Vision $\frac{6}{12}$, $\frac{6}{9}$. Lens opacities. Died acute cardiac failure.

4. Mrs. W. B. S. (41–114) 48 years. 1898. Compound hyperopic astigmatism. Glaucoma 1921, 71 years. Adrenalin dilated pupils. E.P.C. 1926. R. $\frac{6}{18}$, L. $\frac{6}{9}$. Lens changes. Tension ? + to fingers.

5. Mr. E. E. W. (42–160) 55 years. 1898. Symptoms for 5 years. R. $\frac{6}{9}$. L. $\frac{6}{36}$. Tension R. N., L. + 1. Hyperopic. Characteristic cupping. Nasal loss of L. field. R. N. E.P.C. Glaucoma R. 1906. Observed 2 years R. $\frac{6}{9}-$.

6. Mr. J. S. (48–15) 58 years, 1900. Symptoms $2\frac{1}{2}$ years, R. $\frac{6}{9}$, L. Blind. T. R. + ? L. + 2. Observed $1\frac{1}{2}$ years. Eserin. At last examination R. $\frac{6}{9}-$. Hyperopic.

7. Miss R. Van P. (54–121). 12 years, 1902. Compound hyperopic astigmatism. Glaucoma 1921. 33 years. Change in refraction. Cupping. Dilation of pupils by adrenalin. Slightly enlarged blind spots with faint scotoma

T. R. 23, L. 26. V. R. $\frac{6}{9}$. L. $\frac{6}{9}$. E.P.C. After 6 years. T. R. 18 L. 25. Vision unchanged.

8. Mrs. C. J. R. (54–162) 65 years. 1902. Symptoms 1 year. Vision R. $\frac{6}{18}$. L. $\frac{6}{9}$. Myopic. Cupping, extensive nasal loss of field. Eserin. Irregular attendance. Last seen 1902 (4 years later). Blind.

9. Mrs. J. R. (73–76) 49 years. 1907. Symptoms 1 year. Hyperopic. Tension slightly elevated. Characteristic cupping and contraction of field. Vision R. $\frac{6}{9}$. L. Blind. E.P.C. Last seen 1911 (4 years) R. $\frac{6}{12}$ field smaller. T. N. Consultant advised against operation.

10. Miss I. H. (74–18) 57 years. 1907. Symptoms 1 year. Hyperopic. V. R. $\frac{6}{18}$, L. $\frac{6}{24}$. T. + 1 each. R. pupil larger than L. and less active. Cupping. Extensive field loss. E.P.C. Consultant advised against operation. After 3 years. R. $\frac{6}{12}$, L. $\frac{6}{36}$. T. + 2.

11. Mr. D. B. (79–86) 65 years. 1908. Hyperopic. R. Blind 3 years. Recent failure of L.V. L. $\frac{6}{9}-$. T. + 2. Cupping. Nasal loss of field connected to blind spot. E.P.C. 3 years. $\frac{6}{36}-$ T + 2. Rapid loss in 3 months.

12. Miss I. S. R. (82–72) 46 years. 1909. Compound hyperopic astigmatism and presbyopia. Glaucoma 1920. $\frac{6}{9}$ each. T. R. 26 L. 50. Cupping. No pain. Enlarged blind spots and scotomata. E.P.C. 6 years. R. $\frac{6}{9}$, L. $\frac{6}{9}$. Tension L. not controlled, field contracting. Consultant advised operation.

13. Mr. L. W. B. (5338) 54 years. 1917. Compound hyperopic astigmatism. V. R. $\frac{6}{9}$. L. $\frac{6}{9}$. T. 35–40 each. Cupping. Nasal loss of field around fixation connecting with blind spot R. L.N. E.P.C. After 10 years R. $\frac{6}{9}$ L. $\frac{6}{9}+$. T. 25 each.

14. Mrs. E. W. H. (5938) 63 years. 1917. Hyperopic. R. $\frac{6}{36}$, L. $\frac{6}{9}$. T. R. 37 L. 30. Cupping. Contraction of field. After 1 year R. $\frac{6}{18}$ L. $\frac{6}{9}$. T. R. 37 L. 33.

15. Mr. N. M. W. (6059) 73 years. 1917. Hyperopic astigmatism. Symptoms for 6 months. V. R. $\frac{6}{9}$. L. $\frac{6}{9}$. Pupils sluggish. T. R. 37 L. 27. Deep cupping. Extensive nasal loss of field. 1919. $\frac{6}{9}$ each. T. R. 37 L. 30.

16. Mrs. W. H. N. (7610) 51 years. 1919. R. Blind 2 years. L. failing 2 years. V. L. $\frac{6}{18}$. T. R. 80 L. 62.

Deep cupping. Nasal loss of field. E.P.C. After 1 year and 3 months $\frac{6}{18}$. T. 36.

17. Mr. F. H. (7618) 37 years. 1919. R. Myopic astigmatism. L. Hyperopic. V. R. $\frac{6}{12}$, L. $\frac{6}{6}$. Pupils sluggish. T. R. 30. L. 35. Cupping. Loss of nasal field. E. P.C. Iridectomy R. 1921. R. progressively failed 1922. R. $\frac{6}{36}$. L. $\frac{6}{6}$. T. 25 each.

18. Mrs. H. E. T. (8122) 64 years. 1920. Hyperopic. R. failing from glaucoma 15–16 years. Blind 6 months. Enucleated. L. $\frac{6}{6}$ T. 35. E.P.C. After 3 years $\frac{6}{6}$ T. 30, at last examination.

19. Mrs. A. O. (8452) 75 years. 1920. Compound hyperopic astigmatism. Glaucoma 1921. R. $\frac{6}{12}+$ L. $\frac{6}{6}-$. T. R. 22 L. 42. E.P.C. Lens changes. Cupping. 1 year later R. $\frac{6}{9}$.

20. Mrs. E. C. (A–1115) 52 years. 1923. R. failing 5–6 years. No pain. R. blind L. $\frac{6}{9}$. T. 70 each. Cupping. Enlarged B.S. Pupil sluggish. E.P.C. After 4 years $\frac{6}{6}$. T. 26. Hyperopic.

21. Mr. G. C. B. (A–2322) 73 years. 1924. Hyperopic R. $\frac{6}{9}$ L. $\frac{6}{12}$. T. R. 18 L. 37. Nasal loss of field connected with blind spot. Markedly dilated episcleral veins. Cupping with new vessels on disc. Consultant advised no operation. Adrenalin dilated pupils and reduced tension. E.P.C. 1 year later L. blind. 2 years later R. $\frac{6}{6}$ T. 16.

22. Mrs. R. H. C. (A–2461) 64 years. 1925. Myopic. R. $\frac{6}{18}$, L. $\frac{6}{24}$. T. R. 26 L. 36. Retinal hemorrhage. Nasal loss of field. General arteriosclerosis. E.P.C. After 2 years R. $\frac{6}{36}$ L. hand motion. T. R. 26 L. 43.

23. Mr. J. C. F. (52–190) 48 years. 1901. Hyperopic. Glaucoma. 1919. $\frac{6}{9}$ each. T. 35 each. Cupping. Nasal loss of field. E.P.C. General arteriosclerosis. Consultant advised against operation. 3 years later R. Blind L. $\frac{6}{24}$.

24. Mrs. J. H. N. (A–2599) 40 years. 1925. Hyperopic. $\frac{6}{6}$ each. T. R. 35 L. 58 (Gradle). Shallow cupping. Enlarged blind spots no scotomata. Periphery N. E.P.C. Eleven teeth extracted. After 2 years $\frac{6}{6}$ each T. 18 each.

# TRAINING OF THE ADULT BLIND IN GERMANY.[1]

By Miss BETTY HIRSCH,[2] Berlin.

IT is a great privilege to be allowed to speak here tonight, as I know that only scientific questions are taken into consideration in these meetings; but as the work about which I am going to speak has been built up with the help of Professor Silex in Berlin, I hope that you will be a little interested in the results which we have obtained.

I am not going to speak about the education of blind children or blind young people in institutions, as they are almost the same in all countries, and I have seen so much good work done in this direction while traveling around in this country; but I want to speak about the training of our blinded soldiers and blind adult civilians, as the condition of our blind adult persons in Germany has completely changed since the war.

I am often asked by eye doctors to tell how best to prepare grown persons for their future blindness if their cases are hopeless, and as I have had in the past years the opportunity of training hundreds of grown persons in this direction I can speak from experience. But before I pass over to this

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[1] Read before Eye Section, New York Academy of Medicine, November 21, 1927.

[2] Becoming blind at the age of eighteen, Miss Hirsch attended a school for the blind to learn reading and writing Braille, typewriting, music and manual work. Then she went to a college for music amongst seeing people, and studied four languages. Until 1914 she acted as a concert singer and teacher of music and languages. At the beginning of the war she was in London, attending a course at the University of that city, and since her return to Germany in 1914 she has devoted her services to the war blinded.

subject, I want to say a few words about the word "blindness" itself. Do not become sad, because I am not going to tell you anything sad. On the contrary, I want to say that it is a great mistake to look upon blindness as misery. It is of course a handicap, but this handicap can be reduced more than sighted people think if they do not themselves make the blind person unhappy, of course without knowing it, by pitying him instead of learning to understand his position and helping him to feel that he is a normal human being. Seeing persons only too often forget that life, with all of its interests and activities, does not come into us from the outside but that it must be created in our minds and brain. A person with healthy, normal, open eyes can go through life without realizing or comprehending the outer world or life itself, while a blind person usually has a clear picture of life and its contacts impressed upon his mental vision. I think it seems like "carrying coals to Newcastle" when I speak about this subject here in this country where you have a Helen Keller. Another example I would like to mention is our greatest musician of all time, Ludwig von Beethoven, who composed the most divine music when he was perfectly deaf. Did this music come from the outside into his brain, or did he hear it with an inner sense? I think this question answers itself.

If persons who have the gift of sight would realize this they would not find so much difficulty in preparing an adult person for future blindness. Professor Silex, who throughout his life has done so much good for his patients and for blind people, shrank from telling our soldiers the truth about their condition and very often sent them to me; and the task was sometimes not very easy.

After all of the experience which I have had in this direction I am of the opinion that a doctor as soon as he knows that there is no hope of saving eye sight should begin to prepare the patient for his blindness, and not only that, but he should try to get someone to begin the training of Braille and other things, so that the patient may become accustomed to this work before complete blindness sets in. I know from my own experience and from many other cases that this is a great help in such instances.

These were my thoughts when the cruel war began and the blinded soldiers came home from the front. At the beginning of the war I was in England and I stayed there in order to attend a course at the University of London, so I did not get back before the middle of September, 1914. When I arrived in Germany I decided to devote my strength and the rest of my life to these unhappy men who had gone to the war healthy and as free and independent persons, and now came back home broken and blinded and did not know what to do. I found the first five war blinded in the private hospital of Professor Silex, and I went to him and asked if he would allow me to go and cheer his patients up a little and give them instruction in reading and writing. He willingly consented, and on November 22, 1914, I went for the first time to see these men. I found them ready to begin learning, and I taught them reading and writing, Braille, and typewriting. They cheered up very soon, and when this was known in the Ministry of War they sent us for the next few years all of the blinded soldiers who came back from the front. Later on they sent them to their different provinces. So we had throughout the war 600 of them in our school. There are in all 2700 war-blinded in Germany. After they had learned reading and writing they then wished to learn other work and to go into professions and business life again. This created some difficulty, because Professor Silex and I soon realized that we could not put these men into blind institutions, homes or asylums, but the men themselves were a great help to us. We had people of all classes of society, professions and abilities, and we had to separate these into three main divisions. In the first group were men who had excellent education, such as teachers, officers, students and so on. They were instructed in our school in Braille, German and other languages, ordinary typewriting and medical notation, and then they went to the different universities for further study. They graduated, got their degrees and went into physicians' and administration offices. The teachers we got with great difficulty into their former schools for seeing children where they work very successfully. They teach music, chorus singing, history, languages, etc.

The second group were the middle educated people who had been clerks or similar business men and who wanted to get into the business world again. They were trained as typists, stenographers and dictaphone operators. We made some little adjustments on the common typewriters which enabled the blind typists to do the work quite as perfectly as their seeing colleagues. We produced German commercial stenography in Braille and had a stenographic machine made for that purpose. Then there were many men who had been employed by the Post-office. The Postmaster General of Berlin gave us his help in getting these people into the post service again. Some became telephone operators; some did other things, such as folding telegrams, and so on. One of the soldiers himself helped us to a new occupation. He had been a bookbinder before the war and now undertook mending documents. He got along very well, and was engaged in the office of the County Council in Berlin. After his service hours he came to the school and taught his comrades, and now we have eighty document menders in all in the Administration offices in different towns. Securing positions for our typists was very difficult, because of the prejudice of the employers. When we had trained eight typists we invited the Aldermen of the County Council of Berlin to come and see these men work. They were much surprised and praised the work, but when we asked them to employ some of them, one of the Aldermen said that there was not such a law, but another replied, "Then we must have one passed," but the law was not made at that time (that was in 1916). Nevertheless, some days later they sent for two of the typists, and thus came about the introduction of this profession for the blind which is now so generally known in Germany. Then there were in this second division many who had formerly been farmers, and through the kindness of a Count we had for two summers the use of one of his castles, with the grounds, and gardeners who trained the men to do again their former work. Many of them then got a little settlement, with some acres of land which they tilled themselves. We were able to train only ten or twelve people in massage, because the doctors in Germany do not like the blind for this profession; in England there are two hundred

blind masseurs and eighty of the greatest doctors there recommend them to their patients. The few men whom we trained took their examinations amongst the seeing masseurs and secured positions in hospitals.

Then the third and largest division had to be considered. They were people without much education, who had been laborers, factory workers, etc., before they became blind. In this group there was a soldier who gave us an idea. He had been a blacksmith, and he told us that he would never do any work other than connected with iron. I happened to be teaching a little blind girl, the daughter of the Director of our great munitions factories at Spandau, and I asked him to give me the opportunity of trying some work in these factories. I had to get the permission of the Ministry of War, and then I went to the factories, sat down amongst the laborers and tried many of the operations myself. I showed the Directors that it was possible for a blind person to manage the work, and they engaged five of our people. Very soon there were more than twenty or thirty blind workers in all of the different factories in and near Berlin.

This was also the beginning of a new era for civilian blind people. After the war, when millions of men came home from the front to be employed there was great difficulty in finding work for the blind, but out of the more than one million heavily disabled men a union was formed and the men themselves brought before the Government a petition for a law covering the employment of disabled blind. The law was passed in 1919, covering war blinded soldiers. In 1923 there was an amendment of this law, whereby a provision was made for the employment of blind civilians. Afterwards accidentally disabled civilians also came under this law.

Here are a few paragraphs of this law:

All employers who employ at least twenty healthy people are required to employ one heavily disabled. For each fifty healthy employees additional he is required to employ one disabled.

Employers to whom this law refers are private enterprises, corporations and public institutions.

The disabled who come under this law are persons who

are at least 50% handicapped either by war or by accident in civilian life, and those who are blind, deaf and so on.

An employer who does not want to employ a disabled person is allowed to give him a settlement of his own, or to put him in as a tenant.

The General After-care Department for heavily disabled have to work together with the employers in the interest of the disabled. It has to consent if notice is given to a disabled, and this notice has to be given at least four weeks before dismissal.

If an employer violates the requirements of this law he has to pay a fine of from one to one thousand marks. If a disabled individual violates this law the Department can withdraw the advantages of the law from him for a time.

The disabled choose men of confidence, who work together with men of the business staff and the men thus chosen work together with the General After-care Department in the interest of the disabled.

When this law went into effect it was wonderful to see how the blind civilians in Germany awoke to independence and self-reliance. They came to us and asked to be trained in the new vocations, so that they might secure work which would take them out of their former condition, in which they had been dependent on charity, homes and asylums. The Employment Bureau at the Statistical Department in Berlin did splendid work, the result of which you may see by the following table. From 1919 to 1926 war and civilian blind persons were given employment as follows:

| *War Blinded* | *Civilians* | *Nature of Employment* |
|---|---|---|
| 81 | 106 | Metal Industry |
| 4 | 12 | Chemical Industry |
| 0 | 16 | Paper and Cardboard Industry |
| 0 | 5 | Wood Industry |
| 14 | 43 | Food and Victuals |
| 4 | 28 | Textile and Clothing |
| 2 | 11 | Graphical Industry |
| 107 | 52 | Offices |
| 16 | 47 | Professions (three) |
| 6 | 194 | Industries for the Blind |
| 8 | 18 | Mixed Professions |
| Total 242 | 532 | |
| Combined Total.... | 774 | |

This is certainly a good showing when one thinks of the condition of these poor people before the war, and it is a great joy to see how much happier these blind people are who work amongst the seeing, make the same money and are considered as useful members of society.

At the beginning of the war when some of the blinded soldiers came home the Grand Duke Oldenburg introduced the idea of training dogs as guides for the men who were suddenly dependent on their families or friends if they wished to move about. This training has been extended to a high degree, so that now sixteen hundred of our war blinded are guided by dogs, and blind civilians have also asked for these guides. In Oldenburg and in other places in Germany there are special buildings for this purpose; the largest is in Oldenburg itself. There are large kennels for the dogs, grounds filled with all sorts of obstacles and a home for the blind men. Shepherd dogs are brought to these places and seeing trainers do the first work. In about a quarter of a year a dog is trained. The seeing trainer acts like a blind man and tells the dog what to do when coming to an obstacle. For instance, when there is a step the dog sits down and does not move until his master has examined the obstacle with his cane. When there is a lamp-post or anything which might result in an injury if there were a collision the dog draws back and does not go on. If the master drops anything the dog retrieves it and puts it into his hand, and so on. The love of the dog and the blind master for each other is very touching, and the blind man mourns as if he had lost a member of his family when the dog dies or suffers an accident. Many of the blind men can get about only by the help of these dog guides. In the country and in smaller towns the dogs are very satisfactory, but in large cities at street crossings where there is much traffic the men of course are sometimes obliged to ask for the help of other pedestrians. As a whole, however, the dogs serve very well and the men would keenly feel the loss if they were deprived of their faithful companions.

# METHODS FOR PREPARING GROSS AND MICROSCOPIC EYE SPECIMENS.

By Dr. LOUISE H. MEEKER and Dr. WARD H. COOK, New York.

(From the Department of the Laboratories, New York Post-Graduate Medical School and Hospital.)

(*With two illustrations on Text-Plate VII.*)

THIS paper is intended to outline an original method for the preparation of gross eye specimens and a method for microscopic sections that we find most satisfactory. The first has enabled us to augment our teaching equipment and the second to present faithful histological pictures.

## 1. Examination.

The purpose of the laboratory examination of eyes is to study in detail the conditions recognized clinically.

To facilitate the examination in the laboratory the eye should be marked. The marking may be done by the operating surgeon before enucleation, by touching the bulb with silver nitrate near the lesion, or by using thread. Another way is to consult the pathologist and point out the pathological areas as observed in life. This latter plan is the better.

After enucleation the pathologist carefully marks the indicated areas to make certain that they are included in the microscopic sections. This is necessary because certain fixing fluids render the eye opaque at once and greatly obscure the land marks present in the living eye.

## 2. Fixation.

After the eye is marked the next step is fixation so that the picture as seen in life may be preserved unaltered. On no condition should the eye be opened before fixation (freezing is not recommended) for the entire contents of the bulb may escape.

Greeff (1) states in his *Guide to the Microscopic Examination of the Eye:* "The eye in virtue of its peculiar form and composition demands treatment differing in many respects from that sufficient for organs which are more uniform in structure and the precise relation of whose parts is less important to preserve."

The various fixing solutions in common use are unsatisfactory as Verhoeff (4) has pointed out with considerable emphasis. He stresses the frequent artificial detachment of the retina and shrinkage of exudates and ocular tunics after formalin fixation. He states that Zenker's fluid is an uncertain fixing reagent and recommends mixing formalin with a weak alcoholic solution, adding a final word to the effect that this mixture is by no means a perfectly ideal fixing solution.

Greeff (1) nearly thirty years earlier has noted the "great shrinkage of the vitreous and almost always artificial detachment of the retina" when Müller's fluid was the usual fixing reagent for eyes.

We have used Zenker's solution with 5% acetic acid and have added 10% formalin for the fixation of our eye specimens. Formol-Zenker is an old fixing solution but seems not to have been used for eyes. We find that it penetrates quickly, preserves all the ocular structures accurately (Fig. 1) and permits the later application of many histological stains. The solution should be freshly prepared because oxidation takes place rather quickly.

It has been our custom to prepare the fixing solution shortly before the enucleation. The fluid should be about ten times the bulk of the eye, 110*cc.*

Formula:
- Zenker's solution—95*cc*
- Acetic acid (glacial)—5*cc*
- Formalin—10 *cc*

ILLUSTRATING ARTICLE OF DRS. MEEKER AND COOK, "METHODS FOR PREPARING GROSS AND MICROSCOPIC EYE SPECIMENS."

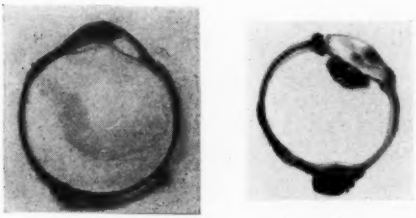

FIG. 1.—Photomicrograph of eye sections fixed by Formol-Zenker. Note the perfect position of the retina.

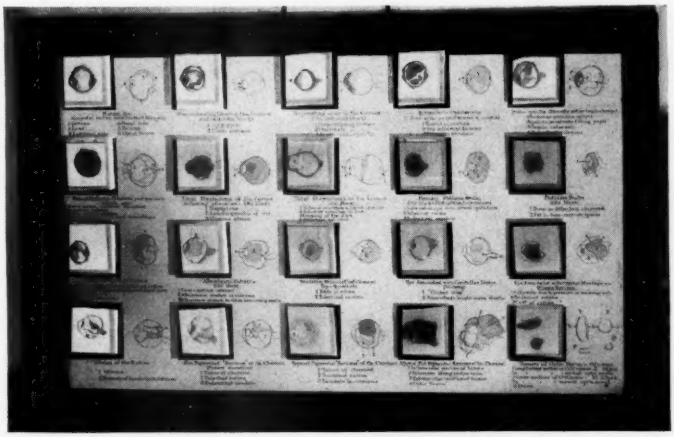

FIG. 2.—The eyes are in museum jars set in a frame. Illumination is transmitted through the jars. Legends are in front beside the jars.

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After the bulb has been immersed in the fixing solution about two hours, small windows are cut in the sclera and the eye replaced in the fixation for 24 hours, then washed in running water 24 hours and placed directly in 80% alcohol. Razor blades are excellent for cutting the sclera. Cut the eye tangentially at opposite poles avoiding the planes to be studied later. Until the sclera is opened the eye always floats in the fixing solution. To insure uniform penetration it is, therefore, well to cover the specimen at first with a thin layer of absorbent cotton.

From now on the usual routine is followed as with any tissue. By our method of fixation the retina remains in proper position, the tissues do not shrink and all elements are well preserved (Fig. 1).

### 3. Sectioning.

After fixation is completed the eye is ready to be cut into sectors as planned from the beginning, that is, the pieces are cut out that are to be embedded in paraffin or celloidin. The cutting is done after the manner described by Greeff (1) and Verhoeff (4). Incisions are made in a plane parallel to the optic axis passing about 0.5*mm* inside the corneal limbus and just missing the lens. It is customary to section the globe in the horizontal plane unless there are lesions best studied in a different plane and to include cornea and papilla in the section with the special lesion to be studied.

The specimens we are showing have been embedded in celloidin by the slow method. After embedding in celloidin the usual microscopic sections are made and stained as may be needed.

Thick sectors are left over uncut and remain embedded in the celloidin. They are placed in 80% alcohol and as a rule either forgotten or thrown away in the course of time. These celloidin blocks become white and opaque and have no apparent value.

We have discovered, however, that these seemingly useless celloidin blocks may be reclaimed and made into excellent gross mounts useful for teaching purposes. This is accomplished by treating with oil so that the celloidin becomes clear

and invisible while the section of the eye remains unaltered. The clearing of specimens is an old process. Spalteholz's (3) clearing method is well known.

Our method makes use of clearing oils following a suggestion in Guyer's (2) *Animal Micrology*. So far as we know we are the first to apply the method to unstained eyes embedded in celloidin for the purpose of making gross mounts.

### 4. Clearing.

The process of clearing may be applied to new celloidin blocks or to old celloidin blocks, even those 20 years old. Several of our most successful preparations are celloidin blocks kept in alcohol about 20 years.

The method is as follows:

Cut block as desired, keeping as thick as possible. Thin blocks may curl and if this happens they must be re-embedded.

1. Place in 95% alcohol 24 hours to remove most of the water.

Subsequent steps admit of two procedures, one short (A) and one long (B).

#### A. Quick Method.

2. Solution { 95% alcohol $\frac{1}{3}$ / Oil of thyme $\frac{2}{3}$ } 24 to 48 hours

3. Solution { Oil of thyme $\frac{2}{3}$ / Castor oil $\frac{1}{3}$

To remain in this until clear (one to several days).

4. Solution { Oil of thyme $\frac{1}{3}$ / Castor oil $\frac{2}{3}$

5. Castor oil.

#### B. Slow Method.

Castor Oil only for days, weeks or months or *until perfectly clear*.

The oil of thyme must be distilled until colorless (one to three times) and be kept in the dark. The castor oil must be as nearly colorless as possible. Baker's cold process "Crystal Castor Oil" approaches whiteness more nearly than any we have been able to procure.

After the clearing process the celloidin is invisible and the eye restored to its appearance before embedding.

This method is not intended to replace other methods of preparing gross mounts of eyes. It is an added method and permits a double use to be made of the celloidin blocks, (1) gross specimens and (2) microscopic sections of the same.

The cleared blocks may now be mounted in castor oil in the usual museum jars for eyes and placed in a special cabinet as pictured (Fig. 2). The blocks are suspended by German silver wires to frames of glass rod. The outlines beside each eye are intended to assist the student to recognize the striking pathology. The eyes are visible from both sides and by proper lighting and some magnification considerable information may be obtained. A background of ground glass gives a pleasing effect when artificial illumination is employed.

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# THE DIVISION OF OPHTHALMOLOGY IN THE NEW UNIVERSITY OF CHICAGO SCHOOL OF MEDICINE.[1]

By Dr. E. V. L. BROWN, Professor of Ophthalmology, Chicago.

*(With one figure in the text.)*

THE aim of the eye clinic is to establish ophthalmology here on a university basis.

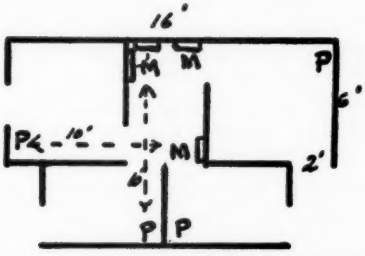
The quarters of the clinic consist of an outpatient department on the second floor, south front, 16.5 by 96 feet, a departmental library staff room and three laboratory rooms adjacent on the first floor, and some ten beds in wards on the third, fourth, and fifth floors. Two additional rooms are held in reserve for eye research on the third floor, north front. Special operating rooms have not been set aside for ophthalmology, but there is ample provision for operative work in the out-patient and general operating rooms.

The out-patient department includes a chief's room, class room, waiting room, clerk's quarters, combined refraction and ophthalmoscopic booths, an external disease room, booths for slit lamp, Gullstrand ophthalmoscopy and a small consultation room. Space for perimetry, tonometry, phorometry, along an adjacent court with good north light, has been loaned to ophthalmology by oto-laryngology. The optician's desk is also located here. The total space thus allocated to the eye clinic, exclusive of ward and operating rooms, is 3,090 square feet, equivalent to a space about 25 by 120 feet.

[1] Read in part at the Formal Opening of the Albert Merritt Billings Hospital and Medical School of the Ogden Graduate School of Science, University of Chicago, October 31, 1927, and offered to this journal for publication as a record of the physical equipment and teaching plans of a new university eye clinic.

The only unusual feature of this arrangement is a group of four inter-locking booths for ophthalmoscopy, retinoscopy and trial case work. They are so arranged that the patient may look through a two foot doorway in one side of the booth into a mirror ten feet away and view the image of the test card placed above his head. The walls of the booths are seven feet high, but made of wood, and extend to a level one foot above the floor. The ceiling above is painted black. Pantasote roller curtains are used in place of doors.

COMBINED OPHTHALMOSCOPIC, RETINOSCOPIC, AND TRIAL-CASE BOOTHS.




P = Patient with test-card above head.
M = 16 x 16 inch mirrors.
SCALE: ½ inch = 1 foot.

The four booths occupy about 150 square feet, or 40 square feet per booth, whereas "twenty foot refraction" requires 90 to 100 square feet per patient. The booths are quiet, airy, no one passes between the patient and the test card and the patient is not moved in the course of his examination with the ophthalmoscope, retinoscope and trial lenses.

I estimate that this space will care for an increase of material up to an average of about twenty new cases per day. For this estimate no existing clinic in this country is of much help, because there is only one with a well salaried personnel, largely on full time, the Wilmer Institute at Johns Hopkins University, and it is not yet in its permanent quarters. Swiss and German eye clinics with twenty new cases a day have about sixty beds, but it is safe to say that the patient "turn-over" in a university eye clinic in this country will

be much greater because conditions such as near-sightedness, eye tuberculosis, and, I believe, wounds and injuries are much less serious problems with us.

## Equipment.

Equipment for study and care of patients has cost about $5,000.00 and for teaching and laboratories, about $6,000.00. Sixteen periodicals and complete back-files of all standard German, French, and English journals and year-books, and many monographs, textbooks have been purchased. Two good eye-libraries have also been presented to the University, one that of the late Francis Lane, Head of the Department of Rush Medical College, so the clinic starts with a good library.

## Funds.

The division has received the following endowments: the Louis B. and Emma M. Kuppenheimer Foundation, $250,000.00; the C. C. Stringer Fund, $250.00; the Gerard von Schaick Foundation, $10,000.00; and in addition shares with other departments in income derived from the Douglas Smith Foundation, $800,000.00. Funds for the conduct of the work are budgeted in two accounts, one "Educational" and the other "Hospital and Dispensary."

Both the hospital and dispensary receive considerable sums from eye patients in limited circumstances but not from eye patients who are able to pay the usual fees. A charge of $3.00 is made for the first visit to all dispensary patients who are able to pay this much, and no more, and $1.50 for each return visit. Refraction cases pay a total of $7.50 for all visits during a period of three months. The clinic makes a moderate profit on all glasses it dispenses.

## Personnel.

The Division is manned by a professor and an assistant professor on part time, and an assistant professor, an instruc-

tor, and staff resident and assistant staff resident on full time, and is provided with a technician and clerk. The salaries paid are respectively, $2,500.00, $1,800.00, $4,000.00, $3,000.00, $900.00, $600.00, $1,500.00, and $1,500.00. A dispensary nurse and a social worker are assigned to the clinic and an optician is in attendance. The assistant resident and resident are each appointed for a period of nine months. It is hoped that a nine months traveling fellowship may be secured for residents who have satisfactorily served the clinic.

### Staff Assignment.

One of the three senior members of the staff is assigned for a period of six months to the care of all cases both medical and surgical, dispensary and hospital. A second senior member is assigned to teaching one hour per day through this same period. The third is free to do investigative work throughout the period. At the end of each six months there is a rotation of assignment. Staff residents assist in all three types of work.

### Teaching.

No one course, even osteology, is required for the title of doctor of medicine,[1] so, as with any other particular course, ophthalmology is elective. A course is offered to a group of six to twenty-five students who meet three mornings a week for twelve weeks. Thirty-six hours of this period consist of a clinical lecture to the entire group. Sixty hours are devoted to a combination of work in eye pathology and ophthalmology (four to sixteen students), twelve hours to a course in methods of examination, and eighteen hours to service as clinical clerks, two to eight in a section,—total of one hundred-twenty-six hours. No post-graduate teaching is to be undertaken in any department at this time.

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[1] "No individual course is specifically required for the M.D. degree." Announcement of "The Medical Schools, 1927-1928, University of Chicago," page 21.

# REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By MR. H. DICKINSON, London.

A Clinical meeting of the Section took place at the Royal London Ophthalmic Hospital (Moorfields) on December 9th, the President, Mr. Ernest Clarke, occupying the chair.

## Hyperphoria Corrected by Operation.

Mr. Williamson-Noble showed this patient, a woman aged 24, who gave a history of severe headache. Her glasses had several times been changed. She had diplopia on awaking, but during the day she got single vision by tilting her head. Prisms gave some relief, but wearing hem was uncomfortable. Vision was $\frac{6}{4}$ on both sides. Neurological examination was negative. With red and green glasses the vertical separation of images was increased on looking downwards. He did recession of left inferior rectus *2mm* under local anæsthesia, and a fortnight later the hyperphoria was corrected to the extent of 1 degree. Headaches had ceased, and unless the patient was lying down and looking over her shoulder there was no diplopia. He described the operation he did in the case.

## Melanosis of Lids and Conjunctiva.

Mr. Cyril Walker showed a case of this condition, and asked whether anything was advised, such as removal of a portion of the melanotic region for microscopy, or whether it

was considered that this interference would quicken the rate of growth. During the 2½ months the case had been under his observation there had been little change, if any. On the conjunctiva the pigmented area looked like a contact product from the original growth.

COLONEL LISTER spoke of a case of his own of similar kind which he had watched 18 months. Opposite to the point of contact was another growth, then it spread round, and became a definite melanotic sarcoma, and he had to remove all the lids and the eye. He recommended removal of a small piece in this case, and if it was malignant it should be dealt with radically.

MISS IDA MANN showed a similar case affecting the conjunctiva, and which had been known to exist 12 years: the patient's age was now 76. It was said to have followed conjunctival ecchymosis. Apparently there had been very little change in the condition over a long period.

### Melanoma of Lid Margin.

MR. MACCALLAN showed a patient with melanoma of the lid margin from whom he had removed the growth. He split the outer canthus wall and then removed all the pigment-bearing conjunctiva, also a V-shaped part from the middle of the lid, leaving a *2mm* margin of unpigmented skin between the growth and his incision.

MR. B. GRAVES thought this was a pigmented mole, as the edges of the tumor did not show signs of malignancy; but a single section was not sufficient for determining whether the growth was malignant.

### Melanotic Sarcoma of Ciliary Body.

THE PRESIDENT showed a section of this condition, remarking that the patient was brought to him with only a pin-point patch on the limbus. The eye was removed, and death ensued five years later from melanotic sarcoma of the liver.

A number of cases for slit-lamp examination were shown by MR. C. GOULDEN and MR. HARRISON BUTLER.

**Relapsed Case of Interstitial Keratitis.**

MR. HUMPHRY NEAME said the man now shown had a definite but not severe attack in the left eye in 1926. Within two months the eye became quiet and vision returned to $\frac{6}{12}$. In the early part of this year a fairly severe attack of keratitis occurred in the fellow eye, the right, and that had now become quiet. But about 6 weeks ago the left eye showed another attack which was very severe, and the patient was suffering from it still.

**Symmetrical Lens Opacities.**

MR. NEAME also showed an instance of this condition in a boy aged 11, the opacities appearing to have been of somewhat recent onset. There was a crescent-shaped opacity in the temporal half of the anterior part of each lens.

**Bilateral Dislocation of Lens.**

MR. M. WHITING showed a brother and sister with this accident; both had small, narrow, pointed heads.

**Obstruction of Macular Artery.**

MR. F. JULER showed a patient with this condition, the second of the kind he had seen in his experience. Diagnostic points of interest were: opacity of the retina, the sudden onset, and alteration in the caliber of the vessel. He did not know the cause, but this woman was three months pregnant. He thought there might be an embolus produced by cells from the placenta; but he had not met with a case of a block of the retinal artery in a pregnant patient before.

**Choroiditis Juxtapapillaris.**

MISS IDA MANN showed this condition in a girl aged 13, who had a slight swelling at the edge of the disc. There was practically no vitreous opacity, and the condition was subsiding. All tests had been negative. The tonsils and

adenoids were operated upon a year ago, and she had a decayed tooth, but nothing else wrong could be found.

### Drusen in a Girl.

MR. D. CARDELL showed a case of this condition in a girl aged 15. He asked whether it was a simple condition, or had a malignant tendency, *i.e.* inflammation of surrounding structures.

### Bilateral Tumor of Orbit.

MR. M. WHITING showed a patient with a swelling in both upper fornices. In the right eye the swelling was large and approached the limbus, and appeared to be fixed to the globe. The tumor seemed to be extending backwards. Wassermann was positive, and a small piece removed from the left tumor had the appearance of lymphangioma. If anti-syphilitic treatment failed, some other treatment would be tried. Lymphosarcoma was rarely bilateral. A blood examination had not yet been done.

### Bilateral Macular Degeneration.

MR. A. C. HUDSON exhibited a girl, aged 7, who until last May had fairly good sight, but it was now very defective. At school it was noticed that she did not look quite straight in front of her. Nothing in her history seemed to explain the condition. It seemed to be akin to the condition described by Mr. Rayner Batten 30 years ago under the name symmetrical macular disease. In neither those cases of Mr. Batten's nor in this patient was there any evidence of disease of the central nervous system.

He also brought a case showing the

### Results on Ante-Natal Corneal Ulceration.

When three weeks old there was a good deal of discharge from the lids of the right eye, but that had now ceased, otherwise the condition was as now seen a month later. Apparently

both corneas were perforated before birth. Both corneas were extensively vascularized at the periphery. At birth the child had an ulcerative dermatitis of the neck, and probably the same toxic agent caused both states.

**Right Superior Temporal Artery Spasm.**

Mr. Humphry Neame showed a case of this condition, which, however, some might label embolism of the artery. A very clean quadrant of the field was absolutely blind. He thought there was still some possibility of recovery of vision.

He also showed a case of

**Retinitis Punctata Albescens.**

There were streaks and spots of pigment, many upon retinal vessels, as in pigmentary degeneration of the retina. There was contraction of the field in both eyes, and night-blindness, hence in some respects there was a resemblance to retinitis pigmentosa.

Mr. Neame's next case was one of

**Macular Degeneration after Retinitis Circinata.**

In 1923 this man had well marked retinitis circinata, and now also had marked angeioid streaks in the retina. Pigment proliferation had occurred, but the streaks were the same. Yet vision in the right eye was $\frac{6}{60}$, and in the left $\frac{6}{36}$.

Mr. Hepburn objected to the term applied to this case, as retinitis circinata was a result of disturbance of the macula; it was an overflow of exudate, some of which organized later.

# DISCUSSION ON OPTIC NEURITIS.

## Section of Ophthalmology, Annual Meeting of the British Medical Association Held at Edinburgh, July, 1927.

By Mr. P. G. DOYNE, London.

Mr. Paterson, in opening the discussion, said that descriptions of morbid appearances at the optic disc had been confused by reason of the variable nomenclature employed. The classification of cases of optic neuritis should be based upon a full investigation of the cases as a whole and not upon any particular observer's idea of the type of disc change present. When cases are studied from a wide aspect, those associated with increased intracranial pressure stand out at once as a class apart. But in early cases of this type it is often difficult to make a diagnosis from the disc appearances, though later on the retinal picture becomes characteristic. The disc changes in this group are almost certainly due directly to the increased intracranial pressure acting mechanically, and the theory that papilledema is caused by the action of irritating or toxic fluid circulating within the nerve sheath is disproved by the speedy disappearance of the discharges after the relief of intracranial pressure. In cases of changes at the disc due to increased intracranial pressure, early decompression gives the best chance of retaining vision.

Mention should be made of cases of optic neuritis following upon severe penetrating head injuries. These were common in the war and were usually associated with sepsis or the

retention of foreign matter. Some of these cases did not appear to be due to increased intracranial pressure.

Turning to cases of optic neuritis definitely not depending upon increased intracranial pressure, speaking generally, these cases show early visual deterioration and a low degree of disc swelling.

There is a group of cases characterized by inflammatory processes developing within the nerve sheath and spreading to the nerve; here syphilis is the most important causative factor.

There is a large group due to toxemia circulating in the blood and giving rise to an inflammatory focus in the nerve. In many of these cases the nerve head is affected later or hardly at all. A central scotoma is the characteristic feature. These cases are really cases of retrobulbar neuritis. In a very large number of such like cases disseminated sclerosis develops after a lapse of some years.

In many cases no satisfactory etiological factor can be established.

With regard to the place of infected posterior nasal sinuses as a causative factor of acute retrobulbar neuritis, Paterson's position is that while many cases of chronic retrobulbar neuritis have arisen from involvement of the nerve in the inflammatory infection of a sinus and while such cases are cured by operation upon the diseased sinus, yet the common acute retrobulbar neuritis has not been proved in the vast majority of cases to be due to spread of inflammation or inflammatory products from the sinus into the nerve.

Dr. Rönne said that the important question to decide was whether the change in the disc is an edema, indicating disease of the brain, or an indication of a local inflammation in the trunk of the optic nerve. The difficulty is that inflammation in the trunk of the optic nerve produces only an edematous condition at the papilla except in those cases where the severe inflammatory process is situated very far forward in the nerve, in which case round cell infiltration may be observed to extend within the eyeball. The one reliable sign of inflammatory papillitis as opposed to papillary edema is the presence of vitreous opacities. It is not possible to go further than to say that high grades of papillary edema are usually associated with increased intracranial pressure. It

is possible, however, to group these cases of retrobulbar neuritis as distinct from the increased intracranial pressure group by means of certain common symptoms:—the central scotoma, the pain in the temples and on movement of the globe, the sensitiveness to pressure and the tendency to recovery.

The pathological anatomy of many of these cases shows a plaque-like formation in the optic nerve, together with destruction of the medullary sheaths of the axis cylinders.

There is a group of cases in which the lesion is almost certainly chiasmal. Here there is a varying ophthalmologic picture from choked disc to normal disc, with general or hemianopic central scotoma combined with hemianopic perepheral defects of the visual field in various combinations. There is a decided tendency to spontaneous recovery in these cases.

MR. BALLANTYNE said that in view of the fact that central vision was affected late in papilledema due to raised intracranial pressure, the early stages of the disc swelling have not been studied very extensively. Choked disc has been defined as a disc with swelling of at least 1.80 diopters but it must begin with slight swelling. He described a case of unilateral papilledema due to a tumor pressing upon the optic nerve which showed the papilledema, and he explains this as being due to direct interference with venous circulation by the growth. He finds in tuberculous meningitis optic neuritis may often be extreme, resembling papilledema. He had observed two cases of encephalitis lethargica with optic neuritis. He recorded a case of papilledema secondary to concussion of the brain, and also a case after severe gastric hemorrhage. With regard to the latter case, the pathogenesis is unexplained. The fact that only one eye was affected places the lesion presumably in front of the chiasma.

With regard to the association of nasal sinus disease with optic neuritis, a cautious attitude should be adopted—so many cases of retrobulbar neuritis get well speedily whatever was done. Many of these cases may later show signs of disseminated sclerosis.

PROFESSOR VAN DER HOUVE advised early operation in papilledema from increased intracranial pressure if vision

was to be preserved. He thought the relative percentages of the several causes of retrobulbar neuritis varied in different countries.

DR. TRAQUAIR suggested the term "plero-cephalic edema" for the swelling of the nerve in increased intracranial pressure. He thought that rhinogenic optic neuritis should be confined to cases with actual inflammatory focus in the papilla.

DR. GEORGE YOUNG described a case of plero-cephalic edema with hemiplegia but no signs of skull fracture after a motor car accident. Trephining over the posterior Kronlein point and opening of the dura gave relief. Papilledema disappeared and full vision was restored.

SIR JOHN PARSONS thought papilledema was sufficient as a name and plero-cephalic edema an unnecessary complication. It was true that early cases of papillitis showed as an edema only at first, but this was the case in the early stages of inflammation everywhere in the body. He emphasized the fact that symptoms of retrobulbar neuritis often preceded signs of disseminated sclerosis by many years.

DR. DE SCHWEINITZ referred to the method of distinguishing choked disc from papillitis in the early stages by noticing the absence or presence of inflammatory cells by the slit lamp. The question of when to decompress in cases of choked disc must be decided by frequent examination of vision and visual fields. He thought infected spheno-ethmoidal disease was a definite etiological factor in retrobulbar neuritis of the chronic type.

DR. WILMER said that with regard to the relation of diseased nasal sinuses and retrobulbar neuritis, X-ray photographs often failed to show pathological conditions in the cells immediately in the neighborhood of the nerve. These cells when infected would cause the disease of the nerve, but it might be almost impossible to deal with them surgically without actually damaging the nerve itself.

SIR WILLIAM LISTER referred to the papilledema associated with concussion. He thought there was nothing characteristic about the appearance. The swelling of the disc was slight. He thought the condition was due to a very slight increase of intracranial pressure.

DR. WILDER said that in some operations upon the nasal sinuses the results were so pronounced and successful that there could be no question of the connection between the sinus and eye condition.

# REPORT OF THE SECTION ON OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE.

By Dr. THOMAS H. JOHNSON, Secretary.

MEETING OF NOVEMBER 21ST, 1927. DR. BERNARD SAMUELS, CHAIRMAN.

Dr. Arnold Knapp presented a case of **traumatic ocular paralysis with retraction movements.**

The patient, sixty years of age, had an ethmoid operation on his left side in June, 1926. He was then seen a few days later by Dr. E. Waldstein, who found a paralysis of the internal rectus on the left side. One year later I saw the patient through the courtesy of Dr. Waldstein, and found the condition the same as it is at present. In the primary position the left eye is deviated outward 40°. In moving both eyes to the right, the left eye comes to a full stop in the median position, when there is a widening of the palpebral fissure and apparently a protrusion of the eye, together with a slight tendency of the eye to go downward. When the eyes are moved to the left the action of the external rectus is entirely one of retraction. The eye is pulled into the orbit and the palpebral fissure gets smaller. The sight and the eye ground in both eyes are normal.

Dr. Knapp thought that the condition could be explained by a separation of the internal rectus tendon, with union of this separation to the inner wall of the orbit by scar tissue, whereby no action of the internal rectus on the eyeball was possible; and through the fibrous band the relaxation of the internal rectus did not follow on attempted contraction of the external rectus, and the external rectus could therefore produce only a retraction movement.

Discussion: Dr. Julius Wolff. I saw this case about a

year ago; it is now as it was then. I think this condition is a counterpart of a picture of Duane's syndrome except that it is reversed. It is hard to explain the complete loss of the internal rectus and the development of the retraction symptom on a traumatic basis and I have misgivings as to this being acquired. I think that the condition existed prior to the injury and that the patient is endeavoring to get compensation. If it is correct that the internal rectus was divided and became attached far back on the globe is it not strange that the eye comes forward when using the internus rather than that it be retracted?

DR. KNAPP. The externus relaxes when the internus acts and so the eye comes forward.

DR. JAMES W. WHITE said that the case had not struck him as being congenital and believed it traumatic. He said he had seen a similar case with complete paralysis of the internal rectus, following an ethmoid operation. There was no retraction in abduction in this case however. The most marked case of retraction that he had ever seen, was in a case where the action of the internus was nil and that of the externus was only slightly less than normal. When the eye was abducted the excursion out was somewhat limited, but the retraction of the globe was at least 5*mm*. These cases of atypical retraction syndrome are not as rare as we sometimes suppose. One gets more retraction with the internus fixed and the externus working, than when the externus is fixed and the internus working. He thought an exploratory operation keeping along the nasal wall was advisable. If the proximal portion of the internus was found of sufficient length, it could be brought through Tenon's capsule and sutured to the globe.

DR. ERNST WALDSTEIN: I had occasion to see this patient 5 days after the sinus operation (ethmoid), when patient showed a rather marked exophthalmos, complete palsy of the left rectus internus and diplopia. On July 1st, there was beginning enophthalmos, complete palsy of internus and externus and retraction of eyeball when trying to abduct. X-ray of orbit in September following a suggestion of Dr. Schoenberg was negative.

DR. LOUISE MEEKER read a paper on **methods for pre-**

**paring gross and microscopic eye specimens** (to be published in full).

DISCUSSION: DR. KNAPP asked why did Dr. Meeker abandon other methods for gross specimens for this one. Dr. Meeker replied that other methods had not been abandoned. This is a salvaging method, the specimens are still in celloidin. It is a method of salvaging old celloidin specimens.

DR. SAMUELS. Many eyes are sent to Mr. Burchell at the New York Eye & Ear Infirmary which are ruined by not being properly fixed. Ophthalmologists should learn eyes require special fixing solutions.

A paper by Miss Betty Hirsch of Berlin, **training of the blind in Germany,** is being published in full in this issue.

DISCUSSION: DR. ELLICE M. ALGER. If there is any one lesson that we must get from Miss Hirsch's address, which we have all enjoyed so much, it is that blindness need not be the overwhelming disaster that most of us consider it, for as she says, "happiness comes from within." The blind often impress the observer as a very happy group in strong contrast to the deaf who have apparently lost a much less essential function.

The soldier blind are a much more favorable group for the educator than the civilian blind. They are mostly young and vigorous adults who have a vast fund of social experience acquired during their "sighted" youth and with a hope and courage that are enhanced by the habits of discipline and coöperation and comradeship that are part of the military life. The civilian blind mostly belong to groups at the extremes of life, the young who have no past and have to build up their ideas of the world laboriously and incorrectly through their other senses and are necessarily timid and distrustful, and the old who may be said to have no future because they have lost their sight at an age when learning seems both difficult and futile.

In America we have had our full share of civilian blind and the things that our educators have done with them are sometimes amazing. Fortunately we came into the great conflict after the period of trench warfare and our war blind were consequently very few compared to those of the Continental Countries.

As Miss Hirsch has so strongly emphasized, the first and perhaps the hardest lesson for the blind to learn is the lesson of courage and confidence, to cast aside his natural timidity and helplessness, to do everything for himself and to depend on himself in every possible way. He seems to get the greatest amount of inspiration from blind teachers who have surmounted the same difficulties and from hearing the accomplishments of other blind people who have become great or successful.

Nothing contributes more to this feeling of self confidence than the consciousness that he looks well and dresses well and "belongs." This would justify a great deal of purely cosmetic surgery which can minimize the disfigurements of disease and injury and make the blind man a more welcome addition to the society of the sighted.

There are many avenues of self support open to the blind but he cannot find them for himself. Society must do that for him by pointing out the promising paths and helping him to make a start. It is perfectly natural for him to utilize the appeal of his helplessness on the sympathy of others and a generation ago the proverbial occupation of the impecunious blind was begging in one form or another, which was both easy and lucrative. A growing self-respect has today carried him to the other extreme till many blind take the greatest pride in competing on absolutely equal terms with the sighted. This can be carried altogether too far. We all find opportunities in love or war or politics or business because we are Scotch or Irish or Catholic or Jew and so make unconscious appeal to groups of our fellow citizens, and the appeal is justified if we make our performance good. I believe the blind man in justice to himself should at least not repulse the instinctive effort of society to help him because of his misfortune, but he should be scrupulously careful to make good so far as in him lies.

The blind man can be taught to make many things, as Miss Hirsch has stated, that are both useful and beautiful but his productive efficiency is only about half that of the sighted worker so that he cannot compete in the open market. In Germany they have solved the problem by laws which compel employers to engage a definite proportion of war

disabled victims. I do not suppose that could be done in America. Over here we can even the balance by giving him a practical monopoly of such things as municipal news stands. We can enable him to compete by finding him a market for his product free from the overhead costs of production and marketing which the sighted man's industry has to carry. In New York we have several institutions which furnish a building and find a market for his products. The question is already rising whether it is right for the Commission for the Blind in one state to carry its selling campaigns into other states. Opportunities for the better educated blind are easier to find and we can all think of individuals who have distinguished themselves in business and politics and law and salesmanship in spite of what seems an insuperable handicap.

The blind man's opportunities for self improvement are widening every day. He belongs to a blind man's club. He hears lectures; the accomplishments of other blind people are always open to him. He reads the standard books and has even a newspaper which he can peruse with his finger tips, while perhaps the greatest gift of all, when it is used for some purpose beside the amusement of the moron, is the radio, with its infinite possibilities of instruction and inspiration. The blind man has his shorthand machine and his typewriter. He can be taught music and public speaking. There is in New York today a theatrical company of blind actors which has been very favorably noticed by the critics.

MR. E. M. VAN CLEVE, Principal, the New York Institute for the Education of the Blind:

"What we have heard from Miss Hirsch is illuminating and impressive. It indicates that intelligence has been used in dealing with the problem of the war blinded men in Germany first and then with the civilian blind. And it is intelligence that is needed to meet the problem of blindness. Many people are so stricken with horror at the onset of blindness that they are rendered incapable of useful service in a time of great need. For such a time was this intelligent and devoted woman raised up and the way in which she handled her problem has approved itself by the results.

Intelligence is always needed in dealing with the coming on of loss of sight, such as was shown by the wise mother of

whom I have heard. Her child, a boy of five, met with an accident which caused the loss of one eye; living on a farm in a remote section, skilled medical assistance was not available and the old story of sympathetic ophthalmia and total blindness was told again. "Now," said his father to the assembled family, "we must all do what we can for Francis to wait on him and protect him." "No," said the mother, "we will not wait on him. Francis must be taught to do things for himself. He must be given the chance to do everything just like the other children." So she set him at helpful tasks, such as bringing in a pail of water, peeling potatoes, and the like, and she made of him a self-reliant, helpful member of the family.

You men of the profession have a great opportunity to teach the parents of the child who is going to be blind, despite all that your skill can do to save him, how to meet the great calamity. Not with repining, not with foolish sentimentality and false sympathy, not with coddling and indulgence should the child who can not be saved from blindness be treated; rather should physician, parents, friends encourage him to face his new life with courage and bend his energies to the solving of his problems. Fortunately you have in the Institute of which I am principal a place where the child may be taught how to succeed in mastering these problems and to grow up into useful citizenship.

Miss Hirsch has shown us the new way of meeting the condition and solving the problem of blindness, the way of courage and the way of intelligence.

## ABSTRACTS.

**Recent Advances in Ophthalmology,** by W. Stewart Duke Elder.[1]

This little book is the third of a series of recent advances in various departments of medicine. Its object, as stated in the preface is "to bring within a small compass and to present in a readily accessible form the research work which has been done within the last few years in ophthalmology and its associated sciences. Its main function is to serve as a connecting link between the standard text book and a bulky and cumbersome periodic literature, to point out the trend of modern thought, and to give some indication of what should be accepted as reliable and what should be regarded with reserve." A book with such a purpose should be greeted with pleasure alike by those who have read a great deal of this periodic literature and are a little uncertain as to just which parts of it constitute "advances," and by those who are not able to keep up with the literature in all languages. How far this purpose is served must depend on the erudition and judgment of the author. The present author's erudition can hardly be doubted after reading this book, and in most of the subjects which he has taken up, an admirable selective judgment seems to have been exercised. His previous training as a physiologist has been of especial value in certain chapters, such as those on "The Nature of Light and on the Retinal Circulation." If some readers find the first part of his chapter on light, which even enters upon the theory of relativity, too theoretical, they will find valuable information in his description of the spectrum, the absorption of light of

[1] Published by P. Blakiston, Son & Co., 1012 Walnut St., Philadelphia, 1927. 337 pages with 4 colored plates and 73 text figures. Price $3.50.

different wave-lengths by the refractive media, and the biological effects of light on the various parts of the eye. Due to absorption by the media, the retina can not be injured by ultra-violet light in short exposures, and if it is theoretically possible that repeated exposures may produce lesions by their abiotic effect, it is far from proven that this effect is responsible for senile degeneration of the macula, as claimed by van der Hoeve. The rays of longer wavelength, however, infra-red and visible rays, can cause damage by their heat effect, as in eclipse blindness or after exposure to the flash of a short circuit.

In the chapter on the accommodation, recent evidence supporting both the Helmholtz and Tscherning theories is summarized, including Graves' observation of an aphakic eye in which the lens capsule could be definitely seen to relax during accommodation, which is in line with the Helmholtz theory. He believes this theory, while true in the main, may have to be modified slightly by the evidence of Tscherning and others. In the chapter on Embryology, the plates for which are from the work of Ida Mann, the question of the origin of the vitreous is discussed, with the conclusion that it contains elements from the ectoderm of the lens, the ectoderm of the optic cup, and the mesoderm of the hyaloid system. The question of the origin of the iris musculature seems to be settled, so that we may consider it to be formed by a modification of the ectoderm. In discussing Congenital Eye Defects and their origin, he takes occasion to bring out the fact that recent investigators, Huxley and Carr-Saunders, Finlay, and Poynter and Allen have not been able to confirm the results of Guyer in producing inherited eye defects, so that this work, which has stirred all biology by its claim to having proved the inheritance of acquired characters, will have to be reconsidered. The defects which Guyer claimed to have produced in rabbits were no different in character or frequency from those found by von Szily and Koyanagi in their families of rabbits with hereditary anomalies in closure of the fetal cleft.

In the chapter on Biomicroscopy the technique of slit-lamp examination by the various methods is described and the most important additions to our knowledge of anatomy and

pathology contributed by it are discussed. The illustrations used are chiefly from the works of Butler and Graves. The technique of slit-lamp examination with ultra-violet light is described. The author considers the mercury vapor lamp which he uses to be preferable for this purpose to the arc-light used by European workers, as in the mercury vapor spectrum the heat rays hardly appear. The advantages of the method are due to the greater differentiations of detail in a nearly monochromatic light and to the fact that the lens becomes fluorescent, so that it may be seen through a cornea opaque to ordinary light. The possibilities of biomicroscopy with polarized light and of spectro-analysis in the living eye as worked out by Koeppe are also described. The author believes that examination of the fundus with the slit-lamp is too difficult to be of much practical value.

The advantages of examination of the fundus by red-free light, for which the mercury vapor lamp is again valuable, are discussed. The nerve fibers are best seen by this light, while for study of the blood vessels, the yellow green light employed by Friedenwald, in which the capillaries are visible, is best. Short sections are devoted to Gonioscopy, or examination of the chamber-angle by the instrument devised by Uribe Tronsoco, radiography of the optic foramen, and radiography of the lacrimal passages by the use of lipiodol.

In his chapters on the "Circulation in the Eye and the Intraocular Fluids," the author is especially at home, and his own valuable work as well as that of others, is recounted. He concludes that the figures of Baillart and Magitot of 25 to 30 as the diastolic and 50 to 70*mm* Hg as the systolic pressure in the retinal arteries are too low, and by a method of inserting a manometer directly into a retinal artery found a pressure of 64 diastolic and 88.5*mm* Hg systolic in the cat. He believes this method to be more reliable than that of Baillart, which involves exerting graduated pressure on the eye with a dynamometer while watching the retinal vessels through the pupil. His own method would seem to be open to the same objection of creating abnormal conditions by opening the eye, and other observers have considered Baillart's method more accurate.

He believes the venous pressure to be slightly above the

intraocular pressure, about 21.5*mm* Hg. Though the capillary pressure cannot be estimated by any method yet devised, it must vary from 55*mm* Hg to a few millimeters above the intra-ocular pressure. After considering the chemistry of the aqueous in the light of recent findings, and considering the divergent opinions of Hamburger and Seidel as to its origin and circulation in the eye, he concludes that the aqueous is a dialysate, the dialysing membrane being the capillary walls. "The fluid percolating the vitreous has within explicable limits, a similar composition. It seems to have a similar origin, and is interchangeable with the aqueous." "The intra-ocular fluids are thus comparable in their origin and metabolism with the tissue fluids elsewhere, and differ from them quantitatively only as a consequence of the relative impermeability of the capillaries of the eye." While a great deal of the fluid exchange takes place by diffusion through the canal of Schlemm, it also occurs through the vascularized tissues of the rest of the eye. The various factors which may be responsible for glaucoma are considered. One of these is swelling of the vitreous, which may be considered as a gel, which Baurmann has found to become more turgid as the blood and tissue fluids become more alkaline. Under treatment, the use of adrenalin and hypertonic solutions is discussed.

In an interesting chapter on "Cataract," the various forms of lens opacity which the slit-lamp has brought to light are described, and the chemistry and metabolism of the lens are discussed. The author concludes that two processes are implicated in the pathogenesis of cataract. 1. The effect of radiant energy of any wave length, acting upon the lens fibers, and rendering them more prone to coagulation by other influences, and 2nd, a derangement of the autoxidation system of the lens, decreasing its content in glutathione, a substance necessary to cell metabolism. The serological properties of the lens protein are discussed, and the fact is brought out that Rötth was unable to confirm the findings of Verhoeff which led him to describe reactions to retained lens protein as phako-anaphylactic endophthalmitis. (A false impression is given by the statement that the reviewer and Steinberg obtained results similar to Verhoeff, since these

findings differed with those of Verhoeff in several important particulars.)

In a chapter on "Sympathetic Ophthalmia," the anaphylactic theory is criticized, and the arguments in favor of an infectious origin are advanced. While no organism can as yet be incriminated, the work of von Szily with the ultramicroscopic herpes virus together with the pathological findings of Fuchs offers evidence pointing to the spread of some infectious agent from one eye to the other by way of the chiasm. The value of the slit-lamp in early diagnosis is emphasized, and modern methods of treatment are summarized.

In a discussion of Phototherapy, the mercury vapor lamp as used by the author, and the arc-light with uvial filter as arranged by Birsch-Hirschfeld are described, and the results of their use in various conditions are briefly stated.

The last chapter deals with the "Neurology of the Eye," and is difficult to abstract. The recent work of Brouwer, Behr, Parsons and others is summarized, and their conclusions are explained by diagrams adapted from their reports. It is one of the most valuable chapters in the book, and the difficult subject matter is presented in a commendably clear manner.

One of the most important features of the book is the series of bibliographies which follow each chapter or section, so that the reader interested in any subject may consult the sources and decide for himself as to the validity of the judgments passed upon them.

SANFORD R. GIFFORD.

IV.—**An Illustrated Guide to the Slit Lamp,** by T. HARRISON BUTLER. Oxford University Press, 1927. Selling in the United States for $9.00. An attractively bound volume in cloth, excellent print, with 158 illustrations, 5 colored plates and 144 pages.

In the introduction a graceful tribute is paid to Professor Vogt and the author says "Almost the whole of our technique and the majority of the discoveries with the slit lamp we owe to Vogt, who has created what amounts to a new science."

A description of the Zeiss apparatus and methods of using it are given. The author prefers the glass topped table with the three footed microscope that slides on the glass top to the mechanical stage with double slide. Many Americans who have used both types prefer the mechanical stage which has many advantages over the other type. In his description of the objectives he uses the old and new obsolete classification. Instead of F.55, Ao, A 2, and A 3, the new objectives are called 2, 3, 4, and 5, so labeled according to their initial magnification.

There are chapters devoted to methods of illumination, normal and abnormal cornea, anterior chamber, iris, lens, retrolental space and vitreous. Also on the effect of operations and injuries upon the eye and the value of the slit lamp in medico-legal cases. In his description of the methods of illumination he uses terms made familiar to us by Graves, and which seem more apt than those in the English translation of Vogt. On page 40 a description of a melanotic carcinoma is given and a drawing of it is shown in plate III. We wonder if this is a clerical error and meant to be melanotic sarcoma.

In a chapter on inflammation the author says "The ability to discover cells in the anterior chamber, the retro-lental

space, and the vitreous is the most valuable feature of the microscopy of the living eye, because it allows us to recognize sympathetic ophthalmitis several days in advance of the appearance of more obvious signs. The discussion of sympathetic ophthalmitis is most interesting and instructive. He states that "the slit lamp affords no support to the existence of a hyaloid membrane" but speaks of floating particles in the retro-lental space, the ease with which they are seen, and of hyphæma in the retro-lental space.

Keratic precipitates are classified and their relative importance discussed. That threads and coagula in the anterior chamber are associated with gonococcal iritis is an observation of Devereux Marshall confirmed by the author. He describes the difference between a decentered lens, ectopia lentis, and a subluxated lens and introduces the term fœtal nucleus in lieu of the "outer embryonic nucleus" as used by Vogt.

That convection currents with floating particles may be seen in clefts in certain cataractous lenses is a most interesting observation shown to the author by Mr. Nicholas Hughes.

The illustrations are excellent and the book makes good reading. It will be most helpful to beginners as well as to those who have had a large experience in the use of the slit lamp. A number of misprints indicates a lack of careful editing.

THOMAS H. JOHNSON.

V.—**A Handbook of Ophthalmology,** by HUMPHREY NEAME and F. A. WILLIAMSON-NOBLE, London. Published by William Wood & Co., New York. Price $4.00. 311 pages, 193 figures. 1927.

This is another book designed for the undergraduate student or the general practitioner. It presents the basic facts of Ophthalmology competently, concisely, and for the most part, clearly. Excellent illustrations and good printing lend the book attractiveness. It must however be said that the volume confines itself to ground which for many years has been more than adequately covered by any one of several similar works.

O. P. PERKINS.

## NOTICE.

THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

December, 1927.

PRELIMINARY NOTICE.

The Annual Congress of the Ophthalmological Society of the United Kingdom will be held in London on Thursday, Friday and Saturday, April 19th, 20th and 21st, 1928. The Subjects for Discussion are:

1. "The Treatment of Irido-Cyclitis with Raised Tension."
   Openers { MR. AFFLECK GREEVES. / MR. W. H. MCMULLEN.
2. "The Diagnosis and Treatment of Inflammatory Affections of the Orbit."
   Openers { MR. A. F. MCCALLAN. / MR. NORMAN PATTERSON.

Members who wish to take part in the discussions or propose to read papers should communicate with

MR. H. P. GIBB,
53, Harley Street, London, W. I.
H. P. GIBB / M. L. HINE } Secretaries.

## INVITATION.

### To Attend the International Ophthalmological Congress to be Held in September, 1929, in Amsterdam and The Hague (Holland).

In the name of the International Council and of the National Committee we have the honor to invite you to take part in the international ophthalmological congress which will be held in Holland in September, 1929.

We beg to point out that members intending to read any paper at the congress should, prior to the 1st of June, 1929, address an extract, containing the principal conclusions, to the secretary Prof. Dr. Zeeman.

No communication will be admitted on any subject, unless the respective extract has been received by Prof. Zeeman not later than on the 1st of June, 1929.

The subscription amounts to dutch Fl. 25.—.

We beg to invite you to apply for the membership as early as possible to the secretary of the National Committee: Prof. Dr. W. P. C. Zeeman, Wilhelmina Hospital, Amsterdam, Holland. Please remit your subscription to the treasurer, Dr. H. M. Roelofsz, Managing Director of the Incassobank, 531 Heerengracht, Amsterdam.

Further communications concerning the meetings, symposia, accommodation, excursions, etc., will be addressed to you in due course.

For the International Council:
J. Van der Hoeve, *Chairman.*
E. Marx, *Secretary.*

For the National Committee:
J. Van der Hoeve, *Chairman.*
W. P. C. Zeeman, *Secretary.*